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ORAL SURGERY
ORAL MEDICINE
and ORAL PATHOLOGY

EDITORIAL

TEACHING SURGICAL TECHNIQUE

GROUP participation at private operations three mornings a week proved helpful last year in making the didactic course more interesting to the

Students operating in animal surgery course at Boston University.

graduate student in oral surgery at Boston University. The actual practical experience gained by the student in such a program, however, is limited to assisting and observing.

This year we have introduced, in addition, a pilot course in animal surgery. This course, which is designed to teach the student surgical principles and techniques, is conducted by Drs. Robert I. Litwin and Morton J. Olin. The students are divided into groups of four, each taking his turn as anesthetist, instrument nurse, surgeon, and assistant.

The animals (dogs) are anesthetized by the continuous intravenous drip method and intubated to facilitate respiration. Fundamental procedures, the art of tissue handling, the identification, isolation, and protection of vital structures, the tying of vessels, and suturing are a few of the principles which are emphasized in the course. Intraoral and, more particularly, extraoral procedures, such as tracheotomies, resections, ostectomies, transosseous wiring, and excision of salivary glands are performed. Operating room techniques are carried out in a relaxed atmosphere in the modern Research Building which has just been completed.

The students' response has been most enthusiastic, as evidenced by the following remarks received in answer to a questionnaire:

—More time should be devoted to this course; preferably, it should run through the entire year rather than through one semester only.

—Most of us consider this course the high point of the week.

—We are gaining a great deal of confidence by working in such a relaxed atmosphere.

—The informal, relaxed atmosphere makes the assimilation of techniques much easier.

We at Boston University believe that such a course will help a great deal in preparing a student for his future hospital work.

K. H. T.

OPERATIVE ORAL SURGERY

ORAL PREMALIGNANCY

Problems of Early Diagnosis

John Hertz, M.D., Stockholm, Sweden

Two things are essential: (1) dentists must be aware of their responsibility and know how to cope with it and (2) public education must open the eyes of the population.

THE role played by the dentist in the early recognition of oral cancer cannot be overestimated. The dentist constitutes the front line in the fight against cancer in this region, the more so since he has the opportunity to observe early clinical lesions and submit them to proper treatment immediately. His early suspicion constitutes the most important step toward eliminating the tragic factor of delay in the treatment of cancer in this location, and he must therefore be familiar with all oral conditions recognized as being premalignant.

The term "premalignancy" refers to changes in the tissues which may assume the character of a malignant growth but which, on the other hand, may remain quiescent for a long time, particularly if they are not exposed to irritation.

The statistics from Memorial Hospital in New York, published by Hayes Martin[11-12] in 1949, lucidly illustrate this vital position held by the dentist, as from 27 to 65 per cent of the patients with oral cancer, depending upon the localization of the lesion, saw their dentists first (Table I).

TABLE I

SITE OF CANCER	TOTAL NUMBER	PER CENT WHO FIRST SOUGHT ADVICE OF DENTIST
Gingiva	114	65
Palate	106	45
Tongue	139	35
Buccal mucosa	113	35
Floor of mouth	125	27
	597	

Memorial Hospital, Head and Neck Clinic, New York, 1949 (Hayes E. Martin).

From the Department of Special Surgery (Professor John Hertz, M.D., Head), Royal School of Dentistry, Stockholm, Sweden.

The histopathologic examinations were performed at the Department of Radiopathology (Professor Lars Santesson, M.D., Head), Karolinska Institutet, Medical School.

Other statistics published by Martin to throw light on the paramount importance of early diagnosis in the prognosis of oral cancer indicate a general five-year cure rate of 20 to 30 per cent (Tables II and III).

TABLE II

PRIMARY SITE OF CANCER	TOTAL NUMBER OF CASES	PROBABILITY OF CANCER RECOGNIZED BY DENTIST		PROBABILITY OF CANCER NOT RECOGNIZED BY DENTIST	
		NUMBER OF CASES	AVERAGE TIME BETWEEN FIRST SEEING DENTIST AND FIRST SEEING PHYSICIAN (WEEKS)	NUMBER OF CASES	AVERAGE TIME BETWEEN FIRST SEEING DENTIST AND FIRST SEEING PHYSICIAN (MONTHS)
Gum	68	21 (31%)	3½	47 (69%)	5
Palate	39	17 (44%)	4	22 (56%)	13
Tongue	34	16 (47%)	4	18 (53%)	5½
Mucosa of cheek	27	14 (52%)	4	13 (48%)	11
Floor of mouth	29	18 (62%)	1	11 (38%)	5
Total	197	86 (44%)	3	111 (56%)	7

Memorial Hospital, Head and Neck Clinic, New York, 1949 (Hayes E. Martin).

Cancer of the lip is not included in this statement, as it has a far more favorable prognosis, the five-year cure rate being 65 per cent. The reason this figure is so much larger is that the lip is more easily accessible for inspection and palpation and, consequently, for an early diagnosis. In later statistics Martin's summary figures have increased to 37.5 per cent.

TABLE III

SITE OF CANCER	PER CENT FIVE-YEAR CURES (SELECTED EARLY CASES)	PER CENT FIVE-YEAR CURES (TOTAL NUMBER OF CASES)
Lip	86	67
Tongue	35	30
Commissura oris	50	19
Buccal mucosa	60	24
Palate	37	30
Gingivae	38	32
Tonsil	40	29

Memorial Hospital, Head and Neck Clinic, New York, 1949 (Hayes E. Martin).

An entirely different picture is obtained, however, from statistics of selected cases which came under treatment at an earlier stage; five-year cures were obtained in 35 to 60 per cent of all the selected early cases and in 85 per cent of the cases of cancer of the lip. The figure in the last column of Table II, which represents the patients who came under treatment at a comparatively late stage, also corresponds to the cases represented in the last column of Table III, for which the prognosis is relatively far poorer. In Martin's latest statistics[14] these summary figures average 57 per cent. The results have been improved practically 100 per cent as a result of the early suspicion and consequent early recognition of lesions as cancer.

Similar statistics illustrating the tragic factor of delay are lacking in Scandinavia.

According to statistics published in 1951 by Nielsen[17] of the Radium Centre in Copenhagen, Denmark, oral cancer is responsible for 2 per cent of all deaths from cancer in Denmark.

Statistics from the Radiumhemmet in Stockholm, Sweden, indicate a five-year cure following treatment of oral cancer in from 25 to 30 per cent of the cases. During the last decade it has been possible to force these figures upward slightly, so that Berven[3] in a tabulation from 1959 was able to report five-year cures in an average of 35.2 per cent of the cases, the extremes being 33.9 per cent and 38.2 per cent. Cancer of the lip is not included in these statistics.

The material presented in the statistics from the Radiumhemmet in Stockholm is also representative of a very large area, including the northern part of Sweden with its huge distances.

As will be seen, the final outcome, according to the Swedish statistics just mentioned, is only slightly superior to that obtained in the unselected cases in the American material and inferior to that obtained in the selected early cases, the discrepancy between this group and the Swedish one being approximately 10 to 15 per cent. As the technical quality of the treatment must, in all probability, be considered equal, this discrepancy must be taken to mean that Sweden is still inferior as far as early diagnosis is concerned. Scandinavian pathologists are very strict in their criteria for the diagnosis of cancer. They hold the opinion that it is better to submit one patient too many than one patient too few to treatment. This probably makes the discrepancy even greater. The average time that passed between the patient's first visit to the dentist and the institution of proper treatment is comparatively long, however, even in Martin's early selected cases, running from one to two weeks. In Sweden, according to Berven, the time that is allowed to pass between the performance of a biopsy and the institution of proper treatment must not exceed forty-eight hours, since it has been emphasized that cancer must be considered an emergency as far as treatment is concerned. In spite of this, it is obvious that in many cases far too much time passes between the patient's first visit to the dentist and the biopsy of the lesion suspected of being malignant. Husted[7] has emphasized the importance of this problem by stating that any change that might possibly be a cancer must be looked upon as being malignant until its nonmalignancy has been established. Much remains to be done to bridge the gap between the best average results and the results that can be obtained when a cancer is suspected and treated very early. This gap can, and consequently *must*, be bridged. Two things are essential for a successful result: (1) dentists must be aware of their responsibility and know how to cope with it and (2) public education must open the eyes of the population.

ETIOLOGY—CARCINOGENIC IRRITANTS

In each case attempts should be made to trace possible carcinogenic irritants as etiological factors. Exogenous as well as endogenous irritants have been sought.

In the oral cavity we are concerned with an *embarras de richesses* of irritants of practically all kinds (Table IV) which are taken to be carcinogenic

active factors proper. The endogenous irritants most frequently act as predisposing factors.

TABLE IV. CARCINOGENIC IRRITANTS

A. *Exogenous*
(Often require a certain degree of predisposition, as demonstrated in experiments with cancer transplantation in man)
1. Known *chemical* carcinogenic irritants
Tar—tobacco
Alcohol
2. *Physical* carcinogenic irritants—chronic irritants of any kind.
1. Mechanical (sharp edges—pressure of prosthesis)
2. Thermal—heat
3. Electric—galvanic currents
4. Irradiations
Ultraviolet light (sunlight)
Ionizing irritation (atom bomb action—x-ray)
3. Pre-existing pathologic conditions in loco

B. *Endogenous*
1. Inflammatory conditions
Syphilis
2. Vitamin deficiency
Vitamin B_2 in connection with sideropenia
3. Hormonal imbalance
Experiments with thiouracil
Estrogenic hormones
4. Age

Proof has not been established that *heredity* is an etiological factor.
Proof has not been established that *viral infections* constitute an etiological factor.

Among the recognized chemical irritants, the etiological significance of tobacco must be considered an established fact, practically speaking. The difficulty in obtaining reliable statistics on this point is due to the fact that so many persons are actually smokers. Martin, however, has demonstrated in a section of the population of New York that 90 per cent of the persons at the cancer age, the average of which is taken to be 56 years, smoked tobacco and that 98 per cent of all patients admitted to Memorial Hospital with oral cancer (that is, practically all of these patients) were smokers—and heavy smokers at that. In this connection, the chewing of tobacco that is always lodged in the same place in the vestibulum should be particularly mentioned. Even the chronic exposure of the oral mucous membrane to heat may act as an etiological factor in the development of oral cancer, and in many cases the effect of smoking presumably depends upon the combination of the effects of the heat and of the substances of the tobacco, in all probability a dibenzpyrene.

Concentrated alcohol must be presumed to play a role similar to that of tobacco, but no reliable statistics are available. Nielsen, of Copenhagen, and Berven, of Stockholm, have indicated that oral carcinoma and, more particularly, carcinoma of the esophagus occur with a striking frequency in members of the catering industry, especially waiters, and there is a definite impression of an etiological relationship.

As physical factors, chronic irritations of any kind have been mentioned. Mechanical irritants occupy an important position in the series of carcinogenic irritants, particularly in the oral cavity.[4] We are here concerned especially

with ragged teeth, sharp overhanging fillings and jacket crowns, and badly fitting prostheses.[4] Cases have also been observed in which cancer developed in the form of a tumor of the gingiva around a carious tooth. It may be mentioned that Nielsen has studied the etiological significance of the galvanic current produced by fillings of various materials; differences in potential of as much as 0.4 volt have been measured, and there is a definite impression that these potential differences may play a fairly important role in this connection.[5]

Pre-existing morbid conditions in loco also have a definite etiological significance in the development of oral cancer. In two of the cases reported in this article periodontal disease with pocket formation was found, and in another case the condition occurred as an epulis-like swelling in a place which it was impossible to keep entirely clean.

Endogenous etiological factors are more difficult to trace. Among these endogenous irritants, chronic inflammatory conditions, particularly syphilis, are assumed to have etiological significance. Martin has produced statistics which demonstrate that 6 per cent of the male population at the cancer age (56 years) have positive Wassermann reactions. In Scandinavia the figures are considerably lower, but still there is a definite statistical difference in that Nielsen's studies indicate that 10 per cent of the patients with oral cancer and 18 per cent of the patients with cancer of the tongue present a positive Wassermann reaction.

Ariboflavinosis, vitamin B_2 deficiency, should also be mentioned among the carcinogenic agents. It is possible that this type of vitamin deficiency exercises its influence by reducing the organism's ability to absorb iron from the intestine. In any case, it is beyond dispute that sideropenia, which is characterized by iron deficiency in the blood serum, is fairly important etiologically in the development of oral cancer (Waldenström[20]).

PATHOLOGY

The most important weapon in early establishment of the proper diagnosis in cases of oral premalignant lesions is an adequate biopsy. Even histologically great difficulties may be encountered, however, in the establishment of the diagnosis. According to Martin, MacComb, and Blady,[16] the presence of some degree of "leukoplakia" in the oral membranes after middle age cannot be considered abnormal, as this simply represents a response of the membranes to mild chronic irritations throughout life. Such leukoplakia must therefore be considered part of the natural aging process, comparable to the almost universal mild senile changes (including keratosis of the skin).

Bernier and Ash[1] have mentioned hyperkeratosis, acanthosis, and epithelial hyperplasia as typical features in oral premalignancy; they also emphasize inflammatory subepithelial exudate. As the most important feature, they point to the "malignant" form of dyskeratosis.

The over-all histopathologic pattern in oral premalignancy is dominated by hyperkeratosis and acanthosis, epithelial hyperplasia, and a downward

proliferation of epithelial buds into the underlying tissue. Generally, a greater or lesser degree of differentiation is evident, even though this may be variable.

The epithelium is irregular, most frequently hyperplastic to a varying degree; it may even present papillomatous formations. On the other hand, it may be slightly atrophic. On the surface there may be a moderate dyskeratosis and parakeratosis. The epithelial cells may present a more or less pronounced nuclear and cellular polymorphism. A varying number of mitotic figures also may be present.

The most important criterion in the evaluation of malignant features, however, is the behavior of the epithelial elements toward the adjacent structures. In the cases under review, a downward proliferation of epithelial elements as smaller or larger strings and buds that projected into the underlying tissue was observed. Furthermore, there was a dissemination of isolated nests and islands of epithelial elements, which were scattered in the underlying tissue. Salley,[19] in his experiments on Syrian hamsters, found apparently independent nests of epithelial cells near the bottom of the epithelium, but adjacent serial sections revealed that they were connected with a large mass of cells. Apart from Salley's statement, no isolated growth of nests of epithelial elements as evidence of oral premalignancy has been mentioned previously in the available literature. In the cases under review these epithelial elements were definitely independent, with no demonstrable connection with the surface epithelium, and in some of the isolated areas there was a tendency toward a concentric or whorl-like formation with an increasing degree of keratinization toward the center.

The border line between the epithelium and the underlying tissue is generally well defined; in some places, however, there may be areas in which no distinct demarcation can be seen. The evaluation of this feature may be difficult, however, because of an inflammatory infiltration.

Martin and associates[16] have emphasized the presence of such an inflammatory infiltration as a typical feature in all cases of oral premalignancy. This requirement is doubtful, however, and inflammatory infiltration is not always present in cases which otherwise present pronounced features indicative of premalignancy.

CASE REPORTS

The present article deals with a limited number of cases which presented particular features of importance in early diagnosis as far as premalignancy or malignancy was concerned. The clinical behavior was insidious, rendering the evaluation of possibly malignant features extremely difficult. Histopathologically, all of the cases presented certain common characteristic features, but different degrees of malignancy varying from none at all to true carcinoma were found. The ten patients, four women and six men, varied in age from 22 to 73 years.

The cases under review have been divided into three different groups (Table V) and the over-all histopathologic pattern has been compared with the clinical features.

TABLE V

HISTOPATHOLOGIC GROUPING	CLINICAL GROUPING				
	ERYTHROPLAKIA-LEUKOPLAKIA	"PERIODONTAL DISEASE"	MUCOUS HYPERPLASIA	EPULIS	ULCER
I. Benign	1 (Case 2)	1 (Case 4)		2 (Cases 1 and 3)	
II. Premalignant	2 (Cases 5 and 6)		1 (Case 7)		
III. Malignant	1 (Case 8)		1 (Case 9)		1 (Case 10)

In *the first group* (Cases 1 to 4) certain characteristic histopathologic features were of diagnostic significance as far as premalignancy was concerned, although they did not allow the definite establishment of such a condition.

In *the second group* (Cases 5 to 7) the histopathologic picture justified the diagnosis of premalignancy.

In *the third group* (Cases 8 to 10) the histopathologic features were basically similar to those in the first two groups, but a malignant tumor (a squamous-cell carcinoma) could also be definitely ascertained, and the very point at which the premalignancy passed into a malignant tumor formation could be definitely established in one of the cases.

CASE 1.—

History and Examination.—A 73-year-old man sought dental advice concerning the removal of a tooth, the upper left second premolar. Several teeth were extracted.

On admission, a slight swelling which was firm and nontender was found in the region of the left lower first premolar (Fig. 1). There was no involvement of the lymph nodes. Routine examination was essentially negative.

Treatment.—In conjunction with the extraction of the lower left first premolar, the swelling was excised after having been isolated from the periosteum.

Pathology.—The piece of tissue was covered by a squamous epithelium in which a pronounced inflammatory infiltration was seen. In places buds of highly differentiated epithelium projecting into the underlying fibrous connective tissue, as well as isolated cords and nests of epithelium, were in evidence. The epithelium was well demarcated and presented only slight cellular polymorphism (Fig. 2).

Outcome.—The postoperative course was uneventful.

Follow-up Examination.—Two years after surgery there was no evidence of a recurrence. The patient now wore a prosthesis in both the upper and the lower parts of the mouth. Behind the molar region in the left side a reddish blue tumor the size of a bean was seen; it was soft and fluctuating, well demarcated, and slightly ulcerated at the top. The tumor was excised, and examination demonstrated isolated nests of epithelial elements in the connective tissue as the most characteristic feature.

CASE 2.—

History and Examination.—A 57-year-old man sought dental advice concerning "periodontal disease." Some teeth had been removed in connection with treatment.

The mucous membrane presented a whitish area at the top of the alveolar process in the region of the lower right second premolar and first molar. Similar whitish areas were revealed in the palate. All the areas were well demarcated.

A biopsy specimen was taken from the region of the lower right second premolar and first molar.

Pathology.—The pieces of mucous membrane were covered by a hyperplastic squamous epithelium. An intraepithelial edema was observed, as well as a slight cellular polymorphism.

Fig. 1.

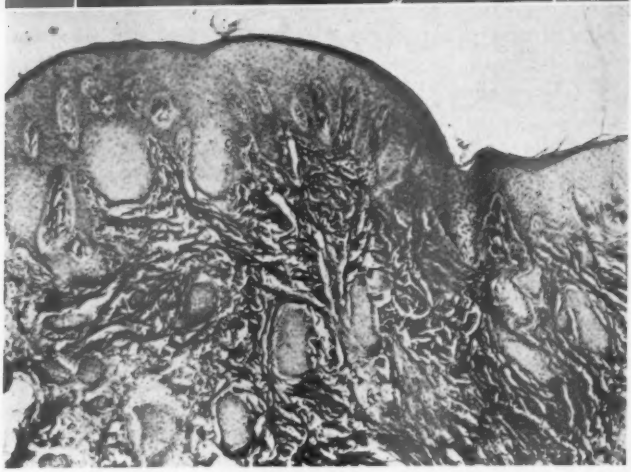

Fig. 2.

Fig. 1.—Case 1. Firm swelling on the gingiva, a so-called "epulis," in which features of diagnostic interest as far as premalignancy is concerned were present histologically.

Fig. 2.—Case 1. Over-all histopathologic pattern of a swelling on the gingiva. Note the epithelial areas projecting into the underlying tissue. There is no marked cellular polymorphism. The epithelium is highly differentiated. (Magnification, ×84; reduced ⅛.)

Fig. 3.—Case 2. Epithelial hyperplasia in "periodontal disease." Occasionally a downgrowth of epithelial buds and strings into the underlying tissue is in evidence (apart from the obviously oblique section of the specimen). The epithelium is hyperplastic, highly differentiated, and well demarcated towards the adjacent tissues. (Magnification, ×84; reduced ⅛.)

In some places a tendency to the formation of epithelial buds and strings was seen. The epithelium was, however, well demarcated toward the underlying connective tissue, in which a slight inflammatory infiltration was seen (Fig. 3).

Outcome.—The patient was feeling well at discharge.

Follow-up examinations were performed at certain intervals. Eighteen months after surgery there were no changes resembling leukoplakia in the mucous membrane on the alveolar process in the region of the lower right second premolar and first molar. A bridge had been made in the region from the cuspid to the third molar in the right mandible following biopsy in the retromolar region. The leukoplakia-like spots in the palatal mucous membrane seemed unchanged.

CASE 3.—

History and Examination.—A 29-year-old woman had a deeply decayed tooth, the upper right second molar removed. Three weeks postoperatively a lump was noticed, growing from the alveolus from which the tooth had been removed. There was no pain or bleeding from the lump. Otherwise the patient was feeling well.

Examination revealed a lump as large as half a bean in the empty alveolus. The lump was reddish, nonulcerated, and nontender. There was no involvement of the lymph nodes. Routine examination was essentially negative.

Treatment.—The lump was surgically removed.

Pathology.—The specimen consisted of granulation tissue covered by a considerable hyperplastic squamous epithelium. The connective tissue was vigorously infiltrated with inflammatory cells. The epithelium was divided into buds and strings growing into the underlying tissue. It presented no pronounced atypia, and no features characteristic of carcinoma were present in spite of the marked tendency to a splitting-up of the epithelial areas (Figs. 4 and 5).

Outcome.—The patient was feeling well at discharge.

Final Outcome.—At follow-up examination one year after surgery there was no evidence of recurrence.

CASE 4.—

History and Examination.—A 22-year-old man was referred by his physician for evaluation and treatment of a condition in the gingiva which caused bleeding, particularly in the frontal region. There had been no acute complications, and the patient had not noticed any loosening of the teeth.

Otherwise, he was feeling well. He knew nothing about morbid conditions. He followed no diet; he was smoking six cigarettes daily; and he was breathing through his mouth.

At presentation the gingiva was reddish in the lower frontal region, with scattered small ulcerations. There was no contact between the front teeth.

A radiogram revealed nothing of significance.

Treatment.—The oral cavity was cleaned and a biopsy specimen was taken from one of the gingival papillae.

Pathology.—The mucous membrane was covered by squamous epithelium with a considerable acanthosis. In a corner of the specimen granulation tissue was found under the epithelium with an abundance of inflammatory cells, between which were numerous plasma cells. The epithelium was split up in some places, and isolated epithelial areas were found in the granulation tissue. The epithelium, however, presented no pronounced polymorphism (Fig. 6).

Outcome.—The postoperative course was uneventful, and the patient was discharged feeling well.

Final Outcome.—Follow-up examination two years after surgery revealed no evidence of a recurrence.

These four cases presented an almost identical over-all histopathologic pattern indicating premalignancy, as it was dominated by an epithelial hyperplasia with a downward projection of epithelial buds and strings as well as

Fig. 4.

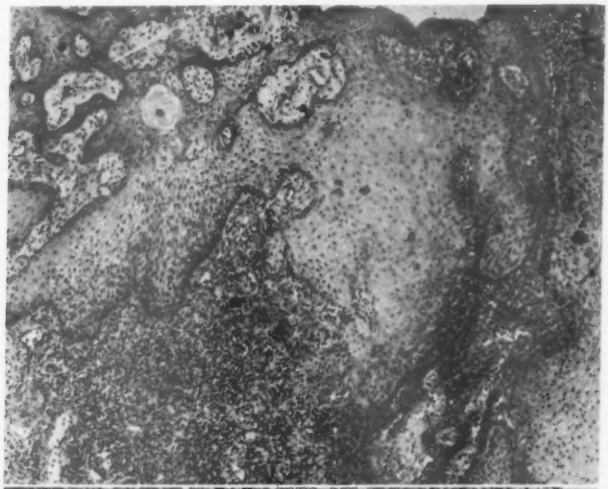

Fig. 5.

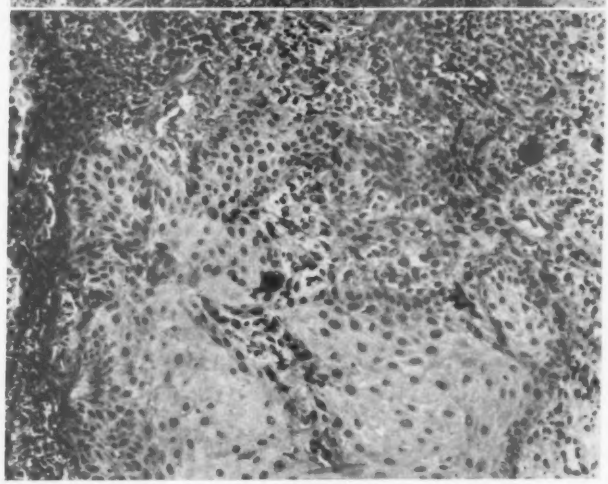

Fig. 4.—Case 3. Granulation-tissue tumor in an empty alveolus after tooth extraction. Note the pronounced hyperplasia of the epithelium with a tendency to the formation of isolated areas in the granulation tissue. (Magnification ×84; reduced ⅓.)

Fig. 5.—Case 3. The epithelium presents no pronounced cellular polymorphism. (Magnification, ×225; reduced ⅓.)

isolated epithelial areas into the underlying connective tissue; these showed a tendency to splitting-up of some of the epithelial areas. Truly premalignant features were lacking, however. The epithelium presented a high degree of

differentiation. There was no pronounced cellular polymorphism or atypia, and the epithelium was well demarcated in all places toward the adjacent structures. In one case there was a considerable acanthosis; in all of the cases there was an inflammatory infiltration under the epithelium.

Clinically, we are faced with very different pictures. In two of the cases (Cases 1 and 3) the lesion presented itself as a firm and nontender swelling—in one case as an epulis on the gingiva and in the other as a lump projecting from an empty socket three weeks after removal of a tooth. In another case (Case 2) the condition appeared as a whitish area, a "leukoplakia," and in the fourth case the lesion presented itself as part of a "periodontal disease."

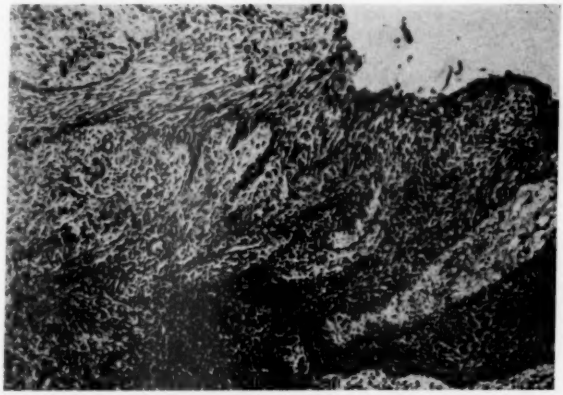

Fig. 6.—Case 4. Biopsy specimen of a gingival papilla from a slight ulcerative gingivitis. Note the splitting-up of the epithelium with isolated epithelial areas in the underlying tissue.

As a *carcinogenic irritant,* poor oral hygiene was noticed in each of the cases; two of the patients (Cases 2 and 4) suffered from a "periodontal disease," and one of them (Case 4) was also a smoker.

Treatment consisted of surgical removal, restoration of perfect oral hygiene, if possible, and elimination of all carcinogenic irritants, including proper treatment of the "periodontal disease" in Cases 2 and 4. After treatment was terminated, the patients were watched carefully.

Prognosis has been favorable; the condition has been brought under control, with no evidence of recurrence during follow-up periods of varying lengths.

CASE 5.—

History and Examination.—A 57-year-old woman sought medical advice concerning a discolored area in the palate. There had been no pain and no sensations from the area, which was noticed accidentally by the dentist who was making a prosthesis. The patient was said to be in good health otherwise. No excessive use of tobacco or alcohol was recorded.

In the palate, corresponding to the prosthesis which had a transverse arch between the hard and the soft parts, there was a reddish, ovoid, nontender area the size and shape of the stone of a date (Fig. 7). The area, which was slightly polypoid along the medial border, was located slightly to the right of the midline. A further proliferation covered by a normal

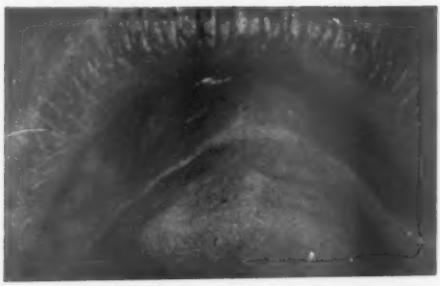

Fig. 7.—Case 5. Innocent-looking reddish area in the palate. Premalignancy was highly suspected as a result of histologic examination.

Fig. 8.

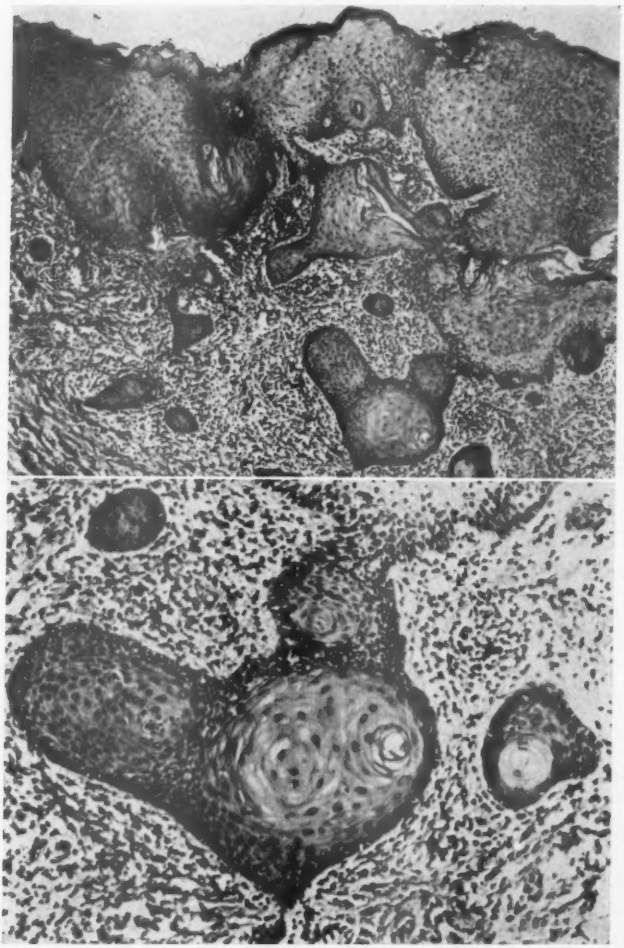

Fig. 9.

Fig. 8.—Case 5. Histologic features indicating premalignancy in an innocent-looking red spot in the palate. The epithelium is highly hyperplastic with projections as well as isolated areas of epithelial elements into the underlying tissue. (Magnification, ×84; reduced ⅓.)

Fig. 9.—Case 5. A comparatively high degree of differentiation is in evidence, and there is only a slight cellular atypia. (Compare with Fig. 8.) (Magnification, ×225; reduced ⅓.)

mucous membrane was found in the front of the arch of the prosthesis. There was no involvement of the lymph nodes. Routine examination was essentially negative. The small proliferation was removed surgically, and a biopsy was performed on a specimen from the reddish area.

Treatment.—The entire reddish area was removed surgically. The patient was forbidden to smoke, and the prosthesis was adjusted. The postoperative course was uneventful.

Pathology.—Microscopic examination revealed that the tissue was covered by a squamous-cell epithelium that was irregular and in some places highly hyperplastic. In some places a growth deep into the underlying connective tissue was seen in the form of buds. These buds, however, presented a comparatively high degree of differentiation, and the cells showed only moderate atypia (Figs. 8 and 9). In the depth, isolated epithelial areas which had a certain tendency to pearl formation in some places were observed. Even in these structures, there was only slight cellular atypia.

Outcome.—The patient was discharged feeling well.

Final Outcome.—Follow-up examinations were performed at certain intervals. Four years after surgery there was no evidence of recurrence.

CASE 6.—

History and Examination.—A 37-year-old man originally sought medical advice concerning a slightly tender swelling in the palate.

Since the age of 5 years he had suffered from a skin disease resembling urticaria, which had been more pronounced in the springtime. He admitted that he had been a heavy smoker, having previously smoked approximately 20 cigarettes daily. A year before, he had ceased smoking. Otherwise he was feeling well.

The swelling in the palate was noticed three weeks before admission and had remained unchanged during this period. The node was removed surgically, and histologic examination revealed that we were concerned with a small polypoid tumor consisting of connective tissue with an inflammatory infiltration and covered by squamous epithelium.

The patient stated that for some time he had been suffering from increasing burning sensations in his left cheek and that he had lately felt such sensations in his right cheek also. A sharp tooth, the lower right second molar, was found and the sensations were ascribed to this tooth, for which reason it was removed. Previously the upper second molars had been removed for the same reason. The condition had had a fluctuating course, with better periods alternating with worse ones. On admission the mucous membrane of both cheeks was covered by reddish and white spots extending over a roundish area, approximately 2 cm. in diameter (Fig. 10). The areas were not sharply outlined; they were not tender, and the mucous membrane was freely movable.

Routine examination was essentially negative. The sedimentation rate was 2 mm. per hour; the hemoglobin was 86 per cent; iron in serum was 120 gamma per cent. Oral hygiene was good. There was no involvement of the lymph nodes.

A biopsy was performed with the extirpation, through an ovoid incision, of the reddish area corresponding to the lower left second molar. A piece extending 2 cm. in depth was excised. The surface was electrocoagulated, and the wound was sutured.

Pathology.—The specimen was covered by a squamous epithelium, which was irregular, in some places hyperplastic and even papillomatous, and in other places slightly atrophic. The epithelium had an increased tendency to proliferation, and areas with a certain cellular atypia were also observed. The underlying tissue presented abundant inflammatory infiltration, and for this reason the demarcation of the epithelium toward the underlying tissue was not well defined in all places.

Treatment.—The entire area involved in the right cheek was removed surgically with no injury to the parotid duct. The wound was undermined and closed. Two weeks later a similar operation was performed on the left side.

Pathology.—The mucous membrane was covered by a squamous, in some parts slightly hyperplastic, epithelium where the cells presented a slight atypia. In the underlying tissue there was a vigorous inflammatory infiltration. The over-all pattern thus presented features similar to those of the piece previously removed (for biopsy) from the other side (Fig. 11).

Fig. 10.

Fig. 11.

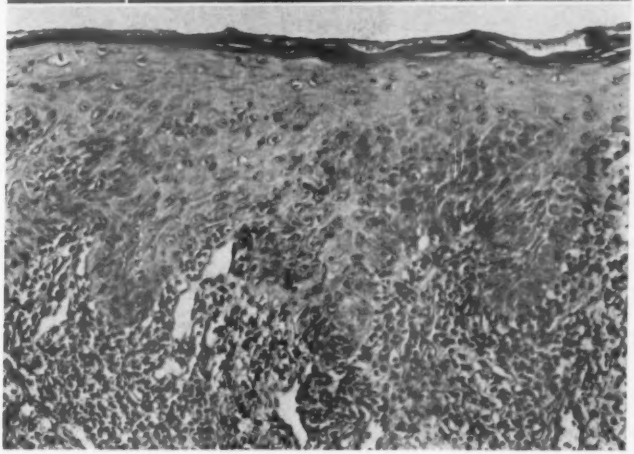

Fig. 10.—Case 6. Erythroplakias and leukoplakias in the cheek with histologic features indicating premalignancy.

Fig. 11.—Case 6. Histologic features indicating premalignancy in an erythroplakia and leukoplakia. There is an obvious proliferation of the epithelium, in some places taking on an irregular character. The borderline between the epithelium and the underlying connective tissue is not well defined in all places. (Magnification, ×225; reduced ⅓.)

Outcome.—The postoperative course was uneventful.

Follow-up Examination.—At follow-up examination one year later a small reddish area in front of the scar in the left cheek was found. It was removed surgically. At follow-up examination after one more year, there was no evidence of recurrence.

CASE 7.—

History and Examination.—A 36-year-old woman sought medical advice concerning a swelling in the palate. The swelling, which was noticed when a new prosthesis was being made, had caused no pain or other discomfort apart from a strange feeling that the patient had when her tongue passed over it. The swelling had increased in size during the two weeks

which had passed after the prosthesis was prepared, for which reason the prosthesis did not fit exactly. One year previously some pathologic condition in the area had probably been noticed by a dentist, as he had inquired about symptoms when correcting the patient's prosthesis.

Fig. 12.

Fig. 13.

Fig. 12.—Case 7. Firm swelling in the palate and reddish spots with histologic suspicion of premalignancy.

Fig. 13.—Case 7. Piece of tissue from a swelling in the palate. The epithelium is hyperplastic and presents a pronounced tendency to proliferation. In some places a marked cellular atypia is observed. Note also the isolated islands of epithelium in the underlying tissues. (Magnification, ×84; reduced ⅓.)

The patient stated that she felt tired and that she had suffered from periodic anemia during the last two years, the hemoglobin percentage having decreased to 68 per cent. She had been treated with injections of liver preparations and iron, which she did not tolerate.

Her mother and her brother died of tuberculosis when the patient was 5 years old.

On admission, a firm swelling, 2 cm. in length and 3 mm. in height, was noticed in the midline in the palate. The swelling, which was nontender and of a slightly reddish color, presented a nodular surface (Fig. 12).

There was no involvement of the lymph nodes, and routine examination was essentially negative. Hemoglobin was 85 per cent, and the sedimentation rate was 4 mm. per hour. The oral hygiene appeared to be good.

A biopsy was performed immediately.

Pathology.—The piece of tissue removed consisted of a mucous membrane with an ulceration and pronounced inflammatory infiltration in the subepithelial connective tissue. The epithelium presented a pronounced hyperplasia with an increased tendency to proliferation; in some places it presented a marked cellular atypia. In the subepithelial tissue, as well as beneath the ulceration, numerous isolated islands of epithelium were found. These areas presented only a slight cellular atypia, however, and they showed a high degree of differentiation (Fig. 13).

Treatment.—The patient was told to have another prosthesis made or to wear no prosthesis at all. She is kept under sharp observation here as well as at the Radiumhemmet.

Follow-up Examination.—Seven months after treatment had started, the area had decreased in size. A slight hemorrhage from the nose had now been noticed, and a minor swelling had appeared in the bottom of the nasal cavity. Two years after treatment there was no evidence of recurrence.

These three cases presented an over-all histopathologic pattern that was basically similar to that found in the four cases in Group I. There were, however, features which indicated a premalignant lesion. Here, too, we found epithelial hyperplasia with a downward projection of epithelial elements as well as dissemination of isolated epithelial areas in the underlying tissues. The epithelium was irregular, with more or less pronounced atypia and polymorphism, and in some places there was a marked keratinization with a tendency to pearl formation, although it presented a comparatively high degree of differentiation. The epithelium was also well demarcated from the adjacent structures, apart from areas in which a vigorous inflammatory infiltration was also present.

Clinically, the behavior was extremely insidious, as the lesions presented themselves as completely innocent-looking, nontender, whitish or reddish spots on the oral mucous membrane or as small proliferations covered by a normal mucous membrane.

As carcinogenic irritants, pressure from a prosthesis had been recorded in two cases (Cases 5 and 7); one of the patients was a heavy smoker (Case 6). In one case (Case 7), a possible endogenic carcinogenic factor was also noticed; for two years the patient had been suffering from periodic anemia which had required injections of liver preparations, since she did not tolerate treatment with iron.

Treatment consisted of the surgical removal of the suspected areas as well as the elimination of all possible carcinogenic irritants, including adjustment or discontinued use of the prostheses. While diagnosis rests primarily with the dentist, proper treatment (particularly in the case of carcinoma) is beyond his capacity; the patient must be referred immediately to a maxillofacial surgeon or a radiotherapist.

Postoperatively, the patient must be carefully watched at regular follow-up examinations.

Prognosis has been favorable, as it is possible to arrest the progression of a premalignancy for many years. In one of the cases (Case 6), a minor recurrent spot had to be removed one year after the first surgical procedure; in another case (Case 5), no evidence of recurrence was noticed during a follow-up period of four years. In no case was hormonal treatment with stilbestrol employed, as it has been in some cases at the Radium Centre in Århus, Denmark.[18]

CASE 8.—

History and Examination.—A 71-year-old man sought medical advice because of a white spot on the left margin of the tongue. He stated that he had noticed this spot for a long time and that it had been growing slowly and steadily. The spot had occasionally caused slight pain.

Fig. 14. Fig. 15.

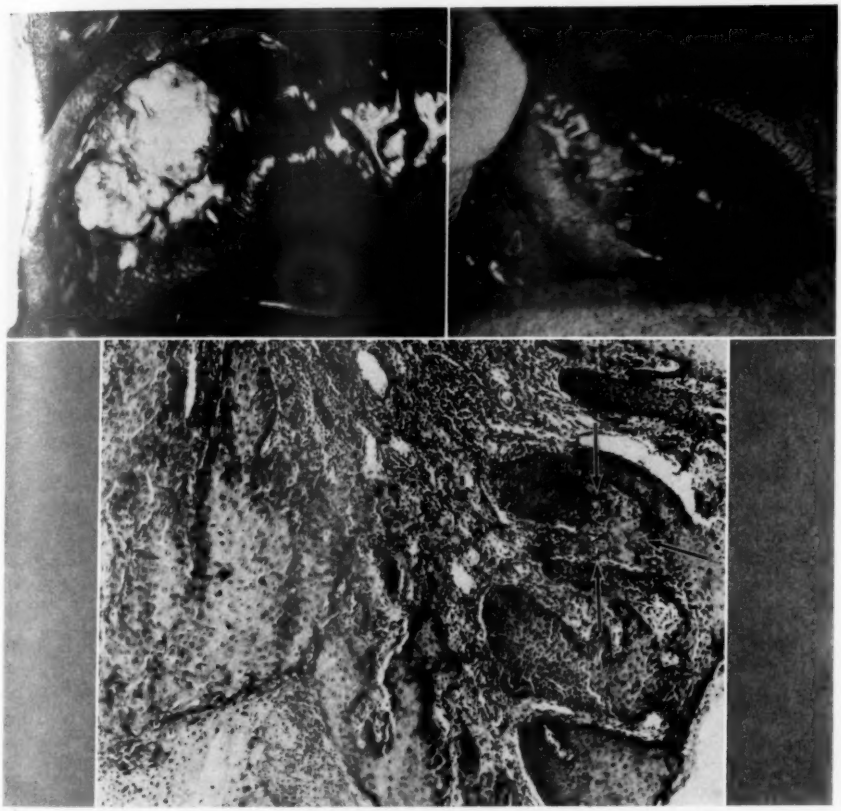

Fig. 16.

Figs. 14 and 15.—Case 8. Leukoplakic area on the margin of the tongue. In one corner the development of carcinoma was established histologically.

Fig. 16.—Case 8. So-called "leukoplakia" with the formation of carcinoma. Note the border between the leukoplakic areas and the actual carcinoma formation. The arrows indicate a place where carcinomatous tissue is scattered among the downgrowing buds and strings from the leukoplakic areas. (Magnification, ×84; reduced ⅓.)

Otherwise, the patient was feeling well. He had formerly been smoking a pipe daily and later two or three cigarettes a day.

On admission, a white, ovoid spot, about 2 cm. long and 1.5 cm. wide (Figs. 14 and 15) was found on the left margin of the tongue, extending down on the inferior side, where the margin was firmer than the more central part of the spot. The consistency was almost the same as that of the adjacent tissues.

Routine examination was essentially negative. There was no involvement of the lymph nodes. The teeth corresponding to the white area on the tongue had sharp edges on the lingual side.

The sedimentation rate was 33 mm. per hour; the blood pressure was 140/100.

Treatment.—I immediately performed a partial hemiglossectomy. The entire discolored area was removed with successive compressing sutures. The hemorrhage was easily brought under control. The patient was given penicillin for five days postoperatively in doses amounting to 600,000 I.U. daily.

Pathology.—The specimen consisted of typically hyperkeratotic and dyskeratotic tissue, with a downgrowth of epithelial buds into the underlying tissue (leukoplakia) (Fig. 16). In one corner a squamous-cell carcinoma presented itself. The degree of differentiation varied. There was slight ulceration, and an abundance of inflammatory cells was seen. As far as could be judged from the specimen, the tumor as well as the entire area which presented the premalignant histopathologic picture had been radically removed.

Outcome.—Aside from a slight edema of the tongue, there were no postoperative complications. The patient was feeling well at discharge and was referred to the Radium Hospital for postoperative irradiation.

Follow-up Examination.—Three years after surgery the patient was feeling well and there was no evidence of recurrence. Four years after surgery he died of heart failure, with no evidence of recurrence.

CASE 9.—

History and Examination.—A 52-year-old woman sought medical advice concerning a plastic surgery procedure to provide a better base for her prosthesis. She had worn the prosthesis for fifteen years. She stated that for some time past she had been suffering from pain in the midline of the upper vestibulum in the region from the left first premolar to the left central incisor. The remnant of a root was found in this area and removed surgically.

One week before admission the patient had experienced pain again, now in the upper vestibulum, and a swelling appeared in the vestibulum. Some discharge was also noticed.

On admission, there was a marked hypertrophy of the mucous membrane on both sides of the frenula in the upper vestibulum. In the lower vestibulum, corresponding to the left first premolar to the left second molar, there was a pronounced frenulum formation.

Routine examination was essentially negative.

Treatment.—The frenula were removed surgically and the wound was sutured.

Pathology.—The mucous membrane was covered by a hyperplastic squamous epithelium at both ends; there was no epithelium corresponding to the middle of it. Along the borders of the specimen, buds were seen growing deeply into the underlying connective tissue. The majority of the buds were solid. There was marked atypia and polymorphism of the epithelium. Deeper in the tissue, several of the buds contained glandlike lumina. Numerous mitoses were seen. There was a vigorous inflammatory infiltration (Fig. 17).

Outcome.—The postoperative course was uneventful. The patient was referred to the Radium Hospital for postoperative irradiation.

Final Outcome.—At follow-up examination one year later, the patient stated that she was feeling well, and there was no evidence of recurrence.

CASE 10.—

History and Examination.—A 51-year-old man sought dental advice because his prosthesis had not been fitting in the right side for the last month. The prosthesis had been worn for a year and had fit well when it was made. There had been some pain during the last three months, radiating toward the right ear, and a swelling had appeared in the vestibulum. The patient had been advised by his dentist, who had noticed an ulceration, not to wear the prosthesis for some time. The symptoms, however, did not disappear in the course of several weeks.

Otherwise the patient was feeling well. He was smoking eight to ten cigarettes a day.

Eight years previously he had been operated upon for a suspected rectal cancer; the condition, however, had proved to be hemorrhoids.

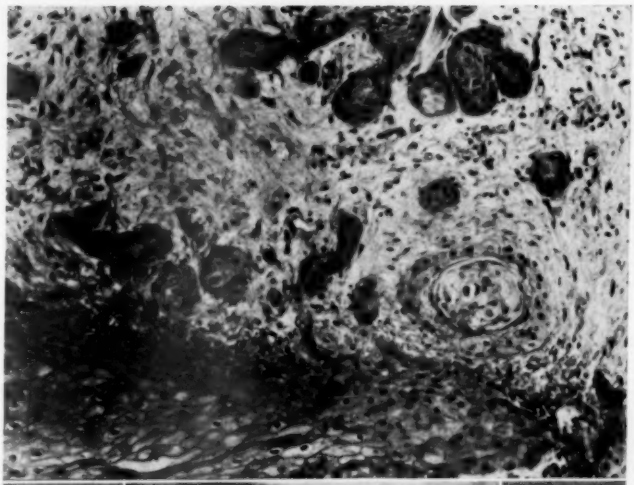

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 17.—Case 9. So-called leukoplakia in an area with the formation of carcinoma. Note the isolated areas of epithelium with a whorl-like arrangement and a tendency to "pearl" formation in one of them. (Magnification, ×225; reduced ⅓.)

Fig. 18—Case 10. Ulcer in the cheek corresponding to the molar region.

Fig. 19.—Case 10. Over-all histopathologic pattern of an ulcer in the cheek presenting definite features of a squamous-cell carcinoma.

TABLE VI. SUMMARY

NO.	SEX	AGE	HISTOLOGIC PICTURE	CLINICAL DIAGNOSIS	POSSIBLE CARCINOGENIC IRRITANT	DURATION
						Group I
1	M	73	Buds of highly differentiated epithelium projecting into underlying tissue	Epulis	Poor oral hygiene	Not stated
2	M	57	Hyperplastic epithelium (leukoplakia); slight cellular polymorphism; epithelial buds and strings were seen; slight inflammatory infiltration; well-defined demarcation	Leukoplakia	Periodontal disease	Not stated
3	F	29	Granulation tissues covered by hyperplastic epithelium; inflammatory infiltration; buds and strings growing into underlying tissue	Granuloma	Occurred 3 weeks after removal of deeply decayed tooth	3 weeks
4	M	22	Acanthotic squamous epithelium, inflammatory infiltration locally; isolated epithelial areas in underlying tissue; no pronounced atypia	Periodontal disease in frontal region	Smoking of 6 cigarettes daily (mouth-breathing); poor oral hygiene	Not stated
						Group II
5	F	57	Squamous epithelium in some places highly hyperplastic; projectings as well as isolated epithelial areas into underlying tissue	Erythroplakia; leukoplakia	Pressure from a prosthesis	Not stated
6	M	37	Irregular squamous epithelium in some places hyperplastic, even papillomatous; increased tendency to proliferation; demarcation of epithelium not well defined in all places	Erythroplakia; leukoplakia	Previously heavy smoking (20 cigarettes daily)	3 weeks
7	F	36	Pronounced epithelial hyperplasia; cellular polymorphism in some places; isolated epithelial areas in underlying tissue	Erythroplakia; hyperplasia of palatal mucous membrane	Pressure from a prosthesis	2 weeks
						Group III
8	M	71	Hyperkeratotic and dyskeratotic epithelium with downgrowth of epithelial buds (leukoplakia); in one corner, squamous-cell carcinoma	Leukoplakia	Smoking	"A long time"

(Continued on

OF TEN CASES

SYMPTOMS	TREATMENT	POSTOPERATIVE COURSE	OUTCOME	FINAL OUTCOME
Group I				
Swelling in the region of $\overline{4}$	Surgery, including extraction and excision	Uneventful	Cured	No evidence of recurrence 2 years after surgery
White spots in region of $\overline{6}$ and $\overline{5}$	Oral hygiene		Unchanged	Unchanged during 18 months
Lump growing from an alveolus 3 weeks after removal of $\overline{7}$	Surgical removal	Uneventful	Cured	No evidence of recurrence 1 year after surgery
Reddish gingiva in lower frontal region with small ulcerations	Oral hygiene		Cured	No evidence of recurrence 2 years after treatment
Group II				
Colored area in palate	Surgery, including total extirpation	Uneventful	Cured	No evidence of recurrence 3 and 4 years after surgery
Burning sensations; reddish and white spots on mucous membrane in both cheeks	Surgery, including total extirpation	Uneventful	Cured	One year after surgery a small reddish area was removed from the left cheek; 1 more year later, no evidence of recurrence
A prosthesis failed to fit in because of a reddish swelling in the palate	A new prosthesis was made			7 months later the area had decreased in size; 2 years after treatment there was no evidence of recurrence
Group III				
White area on left margin of tongue, growing slowly and steadily	Surgical treatment (partial glossectomy); postoperative irradiation	Uneventful	Cured	Died of heart failure 4 years after surgery with no evidence of recurrence

(Continued on following pages.)

TABLE VI

NO.	SEX	AGE	HISTOLOGIC PICTURE	CLINICAL DIAGNOSIS	POSSIBLE CARCINOGENIC IRRITANT	DURATION
9	F	52	Hyperplastic squamous epithelium with buds growing deeply into underlying tissue; marked atypia and polymorphism	Hyperplasia of mucous membrane	Pressure from a prosthesis	"Some time"
10	M	51	Squamous-cell carcinoma	Ulcer in cheek	Pressure from a prosthesis; smoking	3 months

An ulceration was found in the right cheek, corresponding to the molar region. The ulceration, which was not tender, extended even to the soft palate and had a very markedly firm wall (Fig. 18). There was a slight firm nontender swelling of the regional lymph nodes.

Pathology.—Squamous-cell carcinoma was definitely established (Fig. 19).

Outcome.—The patient was hospitalized and submitted to surgery, which included removal of the tumor in the cheek and a right-side neck dissection, followed postoperatively by irradiation.

Shortly after surgery the tumor recurred, and the wound of the neck dissection did not heal posteriorly. X-ray examination revealed metastases to the lungs. The patient died eleven months after he had first consulted his dentist.

In the last three cases the over-all histopathologic pattern was similar to those seen in the cases in Groups I and II, but the condition had the character of a truly malignant tumor, a squamous-cell carcinoma. In one of the cases (Case 8), it was possible to distinguish clearly the place from which the malignant development took its origin.

Clinically, the behavior was extremely insidious, even in the cases which presented symptoms that in all probability would make the patient first see his dentist rather than his physician—in one case (Case 8) on account of an innocent-looking, nontender, white area on the margin of the tongue and in the other cases because a prosthesis did not fit properly.

In another of the cases (Case 9), the dentist found a mucous hyperplasia; in the third case (Case 10) an ulcer was found corresponding to the margin of the prosthesis, which persisted after the prosthesis was adjusted. The most reliable examination in cases of suspected malignancy is palpation, as the most remarkable feature in malignancy is the infiltration growth into the adjacent structures.[7] Even this examination, however, failed in the present cases. Only by biopsy was it possible to establish the proper diagnosis.

As a carcinogenic irritant, the pressure of a prosthesis was recorded. Two of the patients were smokers.

Treatment in two of the cases (Cases 8 and 10) consisted of the radical removal of the whole suspected area, which in one case necessitated subtotal hemiglossectomy and in the other involved even more extensive surgery, including a neck dissection. In the third case (Case 9) only a minor surgical procedure was performed in order to provide a better base for the prosthesis. In all cases, irradiation was given postoperatively at the Radiumhemmet.

TABLE VI (CONT'D)

SYMPTOMS	TREATMENT	POSTOPERATIVE COURSE	OUTCOME	FINAL OUTCOME
Pain and swelling in upper vestibulum in region of 4| to |1	Surgical removal of frenula; pathology established malignancy; referred for irradiation	Uneventful	Cured	No evidence of recurrence 1 year after surgery
Radiating pain; swelling in vestibulum and in right cheek corresponding to molar region	Radical surgery, including neck dissection; postoperative irradiation		Shortly after surgery, tumor recurred	Died of tumor 11 months after surgery

As mentioned in the introduction, the prognosis is far from favorable in cases of oral cancer. Of the three patients reported, one (Case 8) died of heart failure four years after surgery with no evidence of recurrence; another (Case 9) presented no evidence of recurrence one year after surgery; the third patient (Case 10) died of his tumor eleven months after he first went to see his dentist.

SUMMARY

The paramount importance of the role played by the dentist in the early recognition of oral cancer and oral premalignancy, as well as the significance of public education, is strongly emphasized. Statistics are outlined which demonstrate that a great deal remains to be accomplished, as in all probability an increase in the five-year cure rates of approximately 10 to 15 per cent must be considered within reach.

Ten cases are recorded to illustrate the insidious clinical behavior of oral premalignant lesions and the difficulties encountered in attempting to solve the diagnostic riddle. Three groups of conditions are distinguished: one in which the histopathologic picture does not confirm the presence of a premalignant or malignant lesion, although the over-all pattern presents certain histopathologic features that are characteristic from a diagnostic point of view; another in which suspicion of a premalignant condition is justified histopathologically; and a third in which we are faced with a true squamous-cell carcinoma. The highly varying clinical findings are described in each case, and possible carcinogenic irritants, exogenous as well as endogenous, are traced. It is emphasized that it is possible to arrest the progress of a premalignancy by proper treatment, including the removal of all carcinogenic irritants, while arrest of the progress of a carcinoma is still beyond reach.

Two conditions are essential: (1) Dentists must be aware of their responsibility and know how to cope with it. (2) Public education must open the eyes of the population.

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DIET IN ORAL SURGERY

A. E. Nizel, D.M.D., M.S.D., Boston, Mass.

NUTRITIONAL deficiencies are not etiological factors in the types of disease which require oral surgical treatment. Therefore, diet is often overlooked in the over-all management of the oral surgery patient. The dentist should look upon the diet as an adjuvant, similar to analgesics and antibiotics, which is a means of making the patient more comfortable and of speeding recovery. Some of the prime functions of adequate nutrition are (1) to speed convalescence, (2) to promote wound healing, and (3) to increase the patient's resistance to infection.

BASIC CONCEPTS

Animal experiments[8] have shown that the principal nutrients involved in wound healing are protein, vitamin C, riboflavin, and vitamin A. If these nutrients are present in the tissues in adequate amounts, the lag period for restoration of the wound area to maximal strength is shortened. As yet there is no clear-cut evidence that the nutritional factors important to wound healing are the same in man as in animals.[4, 5] However, so much of the nutritional knowledge that has been developed from animal experimentation has been found applicable to man that it seems reasonable to assume that a positive relationship does exist between adequate nutrition and better wound healing.

Increased susceptibility to infection and decreased resistance to established disease may be brought on, in part, by undernutrition,[2] since during a period of undernutrition the ability to manufacture antibodies[9] is decreased. Furthermore, malnourished bone marrow has a decreased capacity to form leukocytes.[1] It has been shown, for example, that protein-deficient animals are more susceptible to infection.[7]

These basic concepts of the role of diet and nutrition in wound healing and in combating infection are only a few of the reasons for the dentist to advise his patient concerning diet. Other reasons are (1) the patient's inability to chew usual foods before tooth extractions because of a broken-down dentition or swelling; (2) his inability to chew normal foods immediately after surgery because of painful and tender tissues; (3) the stress of increased temperature and infection in cellulitis or osteomyelitis which increases tissue requirements for nutrients; and (4) blood loss and excess excretion of nitrogen during extensive oral surgical procedures, which may produce a negative nitrogen balance.

The material presented in this article was first published in a chapter of Dr. Nizel's book, *Nutrition in Clinical Dentistry* (1960, W. B. Saunders Company).

DIET PRIOR TO ORAL SURGICAL PROCEDURES

To assure an adequate tissue-nutrient reserve, the dentist should evaluate the patient's nutritional status before surgery, and particular attention should be paid to the adequacy of protein, ascorbic acid, riboflavin, and vitamin A in the diet. The hematopoietic nutrients, such as iron, vitamin B_{12} and folic acid, deserve special consideration, for their levels may be affected by blood loss from surgery. It is true that in most cases the levels of these hematopoietic nutrients are not seriously affected by routine minor oral surgical procedures. Nevertheless, if the slightest advantage can be gained by provision in the diet of adequate amounts of these nutrients and of those which will promote wound healing, the attempt should be made.

In applying these principles to daily practice, the dentist should start dietary guidance at least one week ahead of time. He should emphasize the importance of eating some complete protein foods, such as eggs, milk, meat, fish, or poultry. Meat (especially the glandular types, such as liver, kidney, and heart), fish, poultry, and eggs not only provide protein but are also rich in iron and the B complex vitamins. Other food sources of these nutrients are green leafy vegetables, whole or enriched cereals, and dried fruits. Liberal use of milk and cheese provides calcium, phosphorus, and vitamin D for bone repair. Patients are prone to omit foods rich in ascorbic acid, which is so important in providing proper intercellular material and collagen for wound healing. For this reason, a pint (two 8 ounce cups) of orange juice (equal to 200 mg. of ascorbic acid or about three times the recommended amount) should be taken daily for at least one week prior to the oral surgical procedure. For the patient with an idiosynerasy to orange juice, there are on the market other fruit-juice combinations which are fortified with vitamin C. Some of these contain acerola cherry juice, which is an exceedingly rich source of vitamin C. Pure ascorbic acid has been added to other fruit juices, such as cranberry juice. If the patient has no difficulty in eating raw fruits and vegetables, such as grapefruit, tomatoes, strawberries, cantaloupe, and green peppers, these foods can be recommended as excellent sources of ascorbic acid.

DIET AFTER ORAL SURGICAL PROCEDURES

One of the first questions that the patient will ask after a tooth extraction or some other oral surgical procedure is, "What can I eat?" Instead of a vague answer, he should be given a specific list of foods or beverages.

If the patient has had a general anesthetic (such as ether) or an intravenous barbiturate which causes a disturbance in fat metabolism, it is best to avoid foods that are high in fat content for the first twelve hours or so after surgery. The patient should be given as much clear fluid as he can tolerate post nausea. In addition to water, such beverages as cola drinks, ginger ale, apple juice, and orange juice, as well as clear broths, are best tolerated. An adequate fluid intake is most important.

When the patient has had oral surgical treatment under local anesthesia, sherbets, junkets, custards, gelatin, and ice cream may be advised in addition

to the above-mentioned beverages. Other suggestions are milk, gruel or cereal, eggs or eggnogs, chicken, pea, or vegetable soup, or a concentrated food supplement, such as Nutrament (12.5 ounces provides 400 calories, 20 Gm. protein, 13.3 Gm. fat, 50 Gm. carbohydrate, some vitamins, and minerals).

The dietary suggestions should be written on the prescription pad along with the other routine postoperative instructions.

To complement the list of foods, an adequate liquid menu which provides variety, balance, and palatability should be suggested for the first day or two postoperatively. This menu is made by selecting foods from the "4 Food Groups" (United States Department of Agriculture Leaflet No. 424, 1958), a simple, convenient guide for providing the dietary allowances recommended by the National Research Council. For example, the patient may have the following foods of each group:

Milk group. Milk in all forms may be used. For extra nourishment, nonfat milk solids may be added to regular milk (3 tablespoons of milk solids to each 8 ounce glass of whole milk). Soft, plain ice creams are also soothing. Milk shakes and malted milks are recommended. Cream may be added to milk if desired.

Meat group. Eggs in the form of strained eggnogs may be used in a liquid diet; eggs may also be used in soft or baked custards. Strained meats (prepared baby food) may be diluted with milk or bouillon to make soups.

Vegetable—fruit group. Citrus juice, such as orange and grapefruit, are highly recommended, as well as tomato juice and other fruit and vegetable juices. Puréed fruits and vegetables, such as baby foods, may be used. Strained fruits may be mixed with milk as a drink, and strained vegetables may be cooked with milk, butter, and other seasonings as soup.

Bread—cereal group. Strained gruels may be used.

A typical day's meals for the first day or two following multiple extractions might be as follows:

Breakfast
Pineapple-grapefruit juice
Strained oatmeal gruel
Coffee (with cream and sugar, if desired)

Midmorning
Orange eggnog

Lunch
Strained cream-of-tomato soup
Custard with cream
Tea or coffee

Midafternoon
Chocolate milk shake

Dinner

Strained pea soup
Spanish cream (or any custard pudding)
Tea or coffee

Bedtime

Cocoa

From the liquid diet the patient can change to a diet of the soft type if and when his soft tissues permit. Such a diet might include the following:

Milk group. Soft cheeses, such as cottage cheese and cream cheese, in addition to milk and ice cream.

Meat group. Tender or ground meats, chicken, fish without bones, and cooked legumes.

Vegetable—fruit group. Cooked or canned fruits and ripe bananas. Vegetables withous skins or seeds; mashed potatoes.

Bread—cereal group. Soft bread, macaroni, noodles, rice, spaghetti, cooked breakfast cereals with milk, and ready-to-eat flaked and puffed cereals, such as wheat or corn flakes and puffed wheat and puffed rice.

Even without being specifically told to do so, the patient will gradually go to the firmer foods as the soreness in his mouth disappears. The important point to stress is that adequate nutrition can easily be maintained if sufficient amounts of foods from the four food groups are included each day.

DIET FOR PATIENTS WITH FRACTURED JAWS

Patients with jaw fractures often suffer other injuries which involve severe blood loss or even shock. For such patients one of the first treatments is the administration of whole blood. After the initial effects of the injury have subsided and the jaw fractures have been immobilized and reduced, feeding becomes a major consideration. The problem involves not only quantity and type of food but also the method of administration.

Before these problems are discussed, some basic principles of the nutrient requirements of this type of patient should be mentioned: (1) At least 2,000 c.c. of fluid should be given to maintain proper electrolyte balance. (2) Protein requirements increase to compensate for (a) loss of nitrogen from injury and (b) tissue repair. (The normal requirement of a healthy man is 1 Gm. of protein per kilogram of body weight, but patients with fractured jaws should have from 1.5 to 2 Gm. of protein per kilogram of body weight if possible.) (3) Adequate calories (in most cases about 2,500) from the other nutrients must be provided daily to spare the protein from being used as an energy provider. (4) If elevation of temperature accompanies the injury, vitamin B complex and C requirements will be increased; therefore, a water-soluble multivitamin capsule of B complex and C in therapeutic dosage should be prescribed.

Parenteral feeding is rarely indicated in patients with fractured jaws and is done only in the event of severe vomiting or other serious complications. In these cases one may administer 5 per cent or 10 per cent dextrose in water with added vitamins plus 5 per cent protein hydrolysate. As soon as practicable, the oral route should be used.

When a patient cannot cooperate, he is fed by tube. Henry and Barrow[3] suggest the use of a small-caliber plastic tube (2.5 mm. or less) which is easily passed through the patient's nose and down into the stomach. In place of the usual prepared liquid foods, Henry and Barrow use natural foods that have been liquefied. Such foods as meats, vegetables, and fruits can be liquefied in a blender and then strained into the fine tube. The liquefied food is then forced through the tube by a mechanical pump. If a mechanical pump is not available, the foods may be forced through the tube by means of an Asepto syringe.

Oral feeding is always preferable to tube feeding. As soon as the patient is able to cooperate, he should be encouraged to take his liquids through a straw or a bent plastic tube. The liquid foods that would be recommended for patients following multiple extractions may also be prescribed for the patient with a wired jaw. Since the patient is likely to be on a liquid diet for several weeks, very careful attention must be given to the adequacy of his diet. The following is a suggested total daily food plan: (1) one quart or more of milk, (2) two eggs, (3) at least one serving of strained meat or chicken, (4) four servings of fruit and vegetable juices (including two citrus juices or juices rich in vitamin C), (5) two or more servings of liquid gruels, and (6) additional calories added via cream and sugars.

In addition to prescribing the proper diet and suggesting ways of serving the necessary foods, the dentist should make certain that the patient actually ingests all the prescribed nutriments. It must be emphasized that food is as important a medicine as an antibiotic or an anodyne. The patient must eat everything that is prescribed for complete and quick recovery.

If he is unable to consume adequate amounts of food, special supplements may be added. If he cannot tolerate or does not relish fruit or vegetable juices, supplements of vitamin B complex and C in therapeutic doses should be prescribed. Protein supplements or oral fat emulsions also may be used if there are indications that the patient needs more nourishment than can be supplied by ordinary foods.

A protein supplement, such as Sustagen, will supply concentrated energy and protein in a small volume; an 8 ounce glass provides 390 calories, 23.5 Gm. of protein, and some vitamins and minerals. One cup of Sustagen can be mixed with an equal amount of water and flavored with ½ teaspoon of vanilla, or 1 teaspoon of instant coffee, or ⅓ cup of orange juice, or 1 tablespoon of cocoa. Another Sustagen recipe calls for 1 cup of milk, 1 egg, 1 teaspoon of vanilla, and 1 large scoop of ice cream. (Measure milk and vanilla into a mixing bowl. Add egg and mix, using rotary egg beater. Add Sustagen and milk and beat until mixture is smooth. Add flavoring. Mix and chill. Add ice cream.) This will yield a 14 ounce glass of fluid which provides 975 calories and 52 Gm. of

protein. Other protein supplements, such as Meritene, can be used in the same way. These are invaluable for the patient with a poor appetite who needs a high protein intake. As soon as the patient's appetite improves, he may discontinue the supplements and receive his nourishment from ordinary foods included in the liquid or soft diet.

Oral fat emulsions have been prescribed for patients who are undernourished and need a concentrated source of calories. Slightly less than ½ cup of an oral fat emulsion supplies 400 calories. For the patient for whom eating is difficult and for whom calories are a prime concern, consideration should be given to the use of oral fat emulsions. Since fat is the most concentrated source of energy, the rationale for oral or intravenous fat emulsions is a sound one, so long as the patient has no untoward reactions. Adequate fat will spare protein from being used as an energy provider.

After the wires have been removed from the jaws, the patient is gradually introduced to solid foods. At first a soft diet, as described in the previous section on diet following multiple extractions, is used. It must be emphasized that the most difficult part of mastication is biting; therefore, the patient should not even attempt to eat such foods as sandwiches, raw apples, and raw vegetables until complete healing has taken place.

DIET FOR PATIENTS WITH CLEFT PALATE

From birth the child with a cleft palate, with or without a cleft lip, presents feeding problems.[6] Early in life he may have to be fed with a medicine dropper or a Brecht feeder, but it is recommended that he be fed from a bottle just as soon as possible. The sucking motions that he uses in extracting milk through a nipple are necessary for the development of the muscles needed for speech and for the development of all mouth tissues.

In an excellent article in which the results of interviews with fifty-eight mothers of children with cleft palate are reported, Zickefoose[10] presents some helpful suggestions for feeding cleft-palate children. Among the suggestions, many of which came from the mothers themselves, are the following:

1. Enlarging the hole in the nipple on the baby's bottle will enable him to get his milk more easily.
2. New nipples may be softened by boiling prior to use.
3. Puréed foods (fruits, vegetables, meats) may be thinned with milk or broth and fed from a bottle that has a nipple with an enlarged hole. (Other mothers found that their children were better able to eat puréed foods that had been thickened with crumbs.)
4. Frequent burping aids in releasing excessive air intake.
5. Eating slowly and taking small bites helps to prevent regurgitation in the older child.
6. The use of a straw helps some children to take liquids more easily.
7. Since feeding a child who has a cleft palate takes longer than feeding a normal child, the mother needs to allow more time for this

task. Fatigue on the part of the parent may interfere with the child's receiving adequate nourishment.

The cleft-palate baby needs the same foods as any other infant. In addition to his milk formula, he needs to have supplements of vitamin D and vitamin C started during the first month of life. He should be given cereals, egg yolk, strained fruits and vegetables, and puréed meats as early as other children. He also should be fed from a cup and spoon at the same time as other children.

Proper diet must be stressed before and after plastic repair of cleft palate. This is particularly important since this surgery is usually performed at the age when the child's appetite is poorest.

DIET FOLLOWING REPAIR OF CLEFT PALATE

Immediately following repair of the cleft, a liquid diet will be provided until healing is complete. The foods to be included should provide sufficient amounts of protein, minerals, and vitamins to promote healing. Liquid foods that will make up an adequate diet were outlined in the early part of this article.

After the cleft has been repaired, the child will be able to eat all kinds of foods. Like any other child, he needs adequate daily amounts of the 4 Food Groups, as follows: milk group, four servings; meat group, two servings; vegetable-fruit group, four servings, including one food that is a rich source of vitamin C and another that is a rich source of vitamin A (at least every other day); and bread-cereal group, four servings of enriched or whole grain.

In addition to the essential foods, a child with a repaired cleft palate should be encouraged to eat foods which require him to use his tongue and his muscles of mastication. Foods that will exercise the muscles used in biting, chewing, swallowing, licking, sucking, and blowing should be provided. Raw fruits and raw vegetables are especially recommended, since they require the use of so many muscles of mastication. Sucking and blowing through a straw or sucking the juice from an orange in which a hole has been cut will provide exercise for the lip and tongue muscles. Licking an ice cream cone is another tongue exercise that can be suggested. Soft cooked fruits, such as apple sauce, also require the use of the tongue. Careful attention to diet will aid in promoting improved speech, good dental health, and improved general health for the child with a cleft palate.

SUMMARY AND CONCLUSIONS

Oral surgical procedures, like those of any other branch of surgery, can disturb body metabolism and upset the nutritional balance and requirements of the patient. Therefore, it is the responsibility of the dentist or oral surgeon to provide his patient with specific advice on nutrition both before and after the surgical procedure. To be meaningful, this advice should include specific foods, meal planning, and menus.

Because protein, ascorbic acid, riboflavin, vitamin A, and the hematopoietic nutrients are essential in promoting wound healing and increasing the patient's

resistance to infection, foods that are rich in these nutrients should be emphasized. When foods from each of the "4 Food Groups" (milk, meat, vegetable-fruit, and bread-cereal) are used, this goal can be attained. Furthermore, liquid or soft diets can be made as adequate as normal diets if foods from the "4 Food Groups" are prescribed for each meal.

Multivitamin capsules, concentrated food supplements, and high-protein supplements are complementary nutritional preparations which should be used as such and not as substitutes for natural foods.

Proper dietary guidance based on a sound knowledge of nutritional principles will promote a state of well-being and make possible rapid convalescence from the stress of surgical procedures.

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26 West St.

ORAL MEDICINE

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EFFECT OF MASSIVE DOSAGE OF A NEW CORTICOSTEROID (TRIAMCINOLONE) ON THE HARD AND SOFT TISSUES OF THE ORAL CAVITY IN THE NORMAL RAT

Edmund Applebaum, D.D.S., Austin H. Kutscher, D.D.S., Edward V. Zegarelli, D.D.S., M.S., and Henry P. Levy, D.D.S., New York, N. Y.

THIS study* was undertaken to ascertain the effect of the administration of large doses of a new, potent, systemically administered corticosteroid, triamcinolone (Kenacort), on the hard and soft tissues of the oral cavity as determined by histologic study of rats treated with this drug (but otherwise essentially intact or normal) during the course of chronic toxicity studies.

MATERIAL AND METHODS

The histologic material that formed the basis of this study was obtained from The Squibb Institute for Medical Research. It consisted of the oral tissues of twenty-four rats that had been used in routine chronic toxicity studies of triamcinolone.

These animals were divided into five groups. Four of the five groups of animals varied only in the size of the dose of triamcinolone administered. The fifth group, which served as the control, was identical in every way except that triamcinolone was not administered. In all other respects (diet, cages, etc.) the animals in all groups were cared for in a similar and routine fashion. The corticosteroid was administered to all animals through tube feeding, hence, its activity on oral tissues was completely the result of a systemic action. The dosage regimens are shown in Table I.

Tissues from four animals in Group I (the highest dosage regimen), one animal in Group II, two animals in Group IV, and three animals in control Group V were studied. Tissues from the remaining animals were available for study if needed.

From the Department of Anatomy of the College of Physicians and Surgeons, Columbia University, and the Division of Stomatology, School of Dental and Oral Surgery, Columbia University.

*The tissues studied in this experiment were obtained from animals which were the subject of chronic toxicity studies at The Squibb Institute for Medical Research.

After the animals were killed, their heads were placed in 10 per cent formalin for at least two weeks. Specimens of the oral mucous membranes were obtained from the cheek, tongue, and lip for histologic study. In addition, block sections of the gingiva, alveolar mandibular bone, and encased first molar tooth were obtained. All soft-tissue specimens were stained with hematoxylin and eosin, while the calcified structures were decalcified and similarly stained.

TABLE I

GROUP	NO. OF ANIMALS	DAILY TRIAMCINOLONE DOSE (MG. PER KG. BODY WEIGHT)	NO. OF DAYS ON WHICH DRUG WAS GIVEN	LENGTH OF EXPERIMENT (DAYS)*
I	4	10.0	74	103
II	5	5.0	74	103
III	5	2.5	74	103
IV	5	0.62	68	97
V	5	2.5 ml. agar per kg.	74	103

*Triamcinolone was not administered to animals on Saturdays and Sundays during this period.

To minimize the effects of such variables as biopsy site, anatomic differences, sectional planes, etc., all tissues were cut and stained in the most exacting and identical fashions possible.

All tissues remained coded (''blind'') throughout handling, processing, and histologic study.

RESULTS

Clinically, the hard and soft tissues of the mouth and jaws of all animals appeared normal at the time of autopsy. Histologic studies of the oral hard and soft tissues did not reveal significant changes in the triamcinolone-treated animals (Groups I, II, and IV) as compared with the control animals (Group V).

DISCUSSION

Triamcinolone has been shown to have markedly greater gluco-corticoid, mineral corticoid, and anti-inflammatory activity than cortisone and hydrocortisone. It has further been demonstrated that some of these actions of triamcinolone may be undesirable in various therapeutic situations in human beings. It was noteworthy, therefore, that no observable clinical or histologic changes were found in the triamcinolone-treated groups of animals studied.

However, the absence of deleterious changes in the oral hard and soft tissues of the triamcinolone-treated rats reported herewith cannot be looked upon as entirely indicative of negative findings for the body as a whole. Nor is it possible to draw proper conclusions as to the effect of triamcinolone thus administered on other areas of the skeleton, since no studies of other organ systems were undertaken in our laboratory.

Of course, extreme care must be taken in the projection of these data (relative to rats) to human patients since species differences are of the greatest

importance in terms of dose response in particular and individual tissue responses in general.

On a weight basis, however, it is obvious that the dosage of triamcinolone was indeed massive when compared to anticipated dosage in human patients. For a 70 kg. man, a triamcinolone dose comparable to that given the Group I animals would be in the neighborhood of 700 mg. per day. The effective dose of triamcinolone in human patients ranges from a maintenance dose of 4 mg. per day to a practical maximum of 30 mg. per day in the management of acute illness.

SUMMARY

The oral mucous membranes, jaws, and surrounding soft tissues of young rats to whom high dosages of a new potent corticosteroid, triamcinolone, had been administered for prolonged periods were studied histologically. All animals were in other respects "intact" and essentially normal.

Comparison of the tissues obtained from triamcinolone-treated and control animals revealed no significant differences in any of the previously normal tissues examined.

The authors express their gratitude to Mrs. Tanya Genningson for technical assistance in the preparation of these tissues for histologic study; to Mr. B. Thomas of The Squibb Institute for Medical Research, under whose direction the chronic toxicity studies of which these animals were a part were conducted; and to Mrs. Eileen Geller and Mrs. Helene W. Kutscher for their assistance.

630 West 168th St.

ORAL MANIFESTATIONS OF HEREDITARY HEMORRHAGIC TELANGIECTASIA

Roy T. Durocher, D.D.S., Alvin L. Morris, D.D.S., Ph.D.,** and Lester W. Burket, D.D.S., M.D.,*** Philadelphia, Pa.*

ALTHOUGH hereditary hemorrhagic telangiectasia is usually referred to as an uncommon disease, Garland and Anning,[1] in an extensive review article, stated that up to 1950 there were 264 families with the disease on record, with approximately 1,500 persons involved. Since that date some fifteen papers[2-16] have been published reporting a total of nineteen additional families, in some of which the disease appeared in many relatives. In 1958, Scopp and Quart[15] published a report which they claimed to be the first in the dental literature of the past forty-five years. Since that time there has been one other.[16] This lack of material in the dental literature is surprising, since, with the exception of the nasal mucosa, the oral mucosa is the most commonly reported site of occurrence.

The occurrence of this disease in the Negro appears to be rare. According to Williams and Brick,[10] no Negro cases were reported until 1948,[17] and since then there have been only two reports[3, 9] involving two Negro families.

Hereditary hemorrhagic telangiectasia is frequently referred to as "Osler's disease," implying that it was first described by Osler.[18] The literature prior to Osler's report reveals that this is not so. It is probable that Sutton,[19] in 1864, in attempting a differential diagnosis of a case of epistaxis with a familial history, was describing hereditary hemorrhagic telangiectasia. It is interesting to note that Sutton observed the teeth and tongue, but made no comment on the oral mucosa proper. He did state, however, that he thought that he observed a skin rash.

In 1896, Rendu[20] differentiated a case of familial hemorrhaging from that of hemophilia. In his discussion of the classic features of the disease, he described epistaxis, a familial history, a general appearance of anemia, small dilated blood vessels or "purpuric" spots of the skin and oral mucous membranes which blanched under pressure, and normal bleeding following the extraction of two teeth. Interestingly enough, although he offered no bibliographic reference, Rendu mentioned that the telangiectases constituted a peculiarity pointed out by one M. Chauffard several months previously.

From the School of Dentistry, University of Pennsylvania.
*Assistant Professor, Department of Oral Medicine.
**Associate Professor, Department of Oral Medicine.
***Professor and Chairman of the Department of Oral Medicine.

When Osler discussed the disease in 1901, he referred to a form of hereditary epistaxis described by Babbington[21] as possibly being similar, but he credited Rendu with first reporting the associated condition of multiple telangiectases of other mucous membranes and of the skin. According to Williams and Brick,[10] Osler described all the organ manifestations recognized today, except the pulmonary angiomatosis. Weber,[22] who with Rendu and Osler, is frequently associated with the disease, apparently did nothing more than point out that the condition gets worse with age and that there is usually the vicious cycle of hemorrhage—anemia—hemorrhage. It was Hanes,[23] in 1909, who singled out the consistent diagnostic triad and termed the disease "hereditary hemorrhagic telangiectasia." In addition to Garland and Anning,[1] Barrock,[24] Goldstein,[25] and Williams and Brick[10] have published extensive reviews of this disease.

Within recent years three investigations[1, 8, 12] of the hereditary nature of the condition have been published. Bird and associates[12] reported on a most extensive and interesting study. The investigators were invited to a family reunion of 170 blood relatives, 19 per cent of whom they later discovered to be afflicted. Twelve per cent of the patients reported hemorrhage from the mouth, and lesions were observed on the lips of 70 per cent and on the tongues of 40 per cent.

The disease is transmitted as a simple dominant characteristic with equal involvement in both sexes. There is some question as to the reason for what seems to be an occasional absence in some generations. Some attribute this to atavism, while others cite the probability that unobservable lesions exist but without hemorrhagic episodes.

Hereditary hemorrhagic telangiectasia is the result of a vascular anomaly. According to Dolowitz,[5] histologic studies reveal that the dilated vessels are thin-walled and characterized by a lack of organization into well-defined coats. Dolowitz states that whether or not elastic tissue is present is a matter of debate, but one of the most recent papers[13] on the disease indicates the absence of an elastic and muscle sheath, with only the endothelial layer remaining. The disease is not associated with any derangement of bleeding time, clotting time, clot retraction, or platelet count.

Hereditary hemorrhagic telangiectasia is characterized clinically by numerous localized dilatations of capillaries, small arterioles, and small venules on the skin and mucous membranes. They are perhaps most frequently found about the face and on nasal and oral mucous membranes, the order of involvement usually being the nasal mucosa, the tongue, the palate, the lips, the mucocutaneous junction, and the gingiva. Other organs, such as the stomach, lung, liver, bladder, and brain, can be involved. The lesions, which often are not apparent until the second decade or even later in life, are bright red, purple, or violet, and range in size from pinpoint to several millimeters in diameter. Sometimes spiderlike configurations are seen, particularly in elderly persons.

Nontraumatic hemorrhage of the telangiectases is a typical clinical feature, and the bleeding becomes more severe and more frequent with age. Some think that the hemorrhages are aggravated by the anemia which they themselves cause,

thus producing a vicious cycle.[10, 22, 23] A most significant clinical diagnostic test is the application of pressure with a glass slide. Partial or complete blanching of the lesion, with a return of the color when pressure is released, indicates that the blood is within dilated vessels.

There is no generally accepted therapy for the telangiectases. Topical hemostatic agents and electrocoagulation have been effective in the control of accessible hemorrhages.[26]

CASE REPORT

A 59-year-old Negro woman came on Jan. 7, 1959, to the Oral Diagnosis Clinic of the School of Dentistry, University of Pennsylvania, to request complete dentures.

The patient was lethargic, apprehensive, and somewhat pale, with purpura-like areas of small but varying size on the facial skin and fingertips. Upon being questioned, she reported that she had been hospitalized for blood transfusions four days prior to this clinic visit.

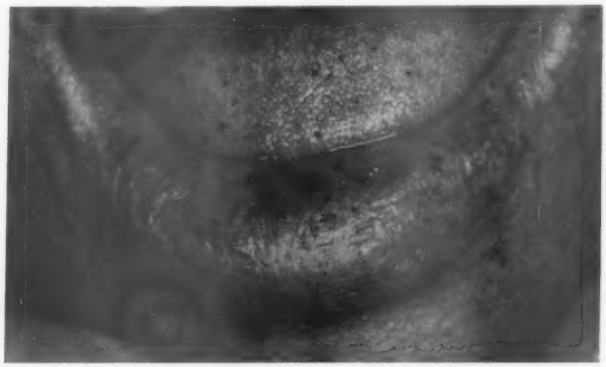

Fig. 1.—Telangiectases on lower lip and dorsum of tongue.

Further investigation disclosed that in January, 1956, the patient presented herself to the hospital of the University of Pennsylvania with the chief complaint of weakness and loss of blood from epistaxis. Her difficulty dated back to some two years following menarche when she began to have frequent nosebleeds. However, she had considered herself in excellent health until the 1940's, when during the menopause she gradually developed weakness, fatigue, and a general lack of pep. When seen by her family physician at that time, she was found to be anemic. This condition was attributed to poor dental hygiene, and following dental extractions and the administration of massive doses of iron and vitamins her strength was restored. Between 1945 and 1954, at intervals varying from six weeks to several months, she would gradually return to a weakened state. Anemia was always diagnosed, and the desired response was obtained with liver injections, iron, vitamins, and, on several occasions, blood transfusions.

In the summer of 1954 telangiectatic areas on the oral mucous membranes were noted for the first time, and their correct diagnosis established the etiology of the anemia. Gastroscopic and proctoscopic examination revealed similar areas involving the mucous membranes of the stomach and sigmoid colon.

When she was admitted to the hospital in 1956, the patient had a hemoglobin level of 5.4 grams per 100 c.c., blood pressure of 110/68, a pulse rate of 72, a temperature of 102° F.,

and a positive guaiac reaction to fecal residue. Except for poor dental hygiene and choroiditis, the remainder of the physical examination was within normal limits. Following blood transfusions and further supportive treatment, the remainder of the patient's teeth were extracted. She has since been hospitalized three times for blood transfusions and supportive treatment.

Family history revealed that, of the patient's seven children, two have telangiectasia of the lips and four are refractorily anemic. Also, the son of a daughter has anemia, although its origin is undetermined and no lesions have been noted.

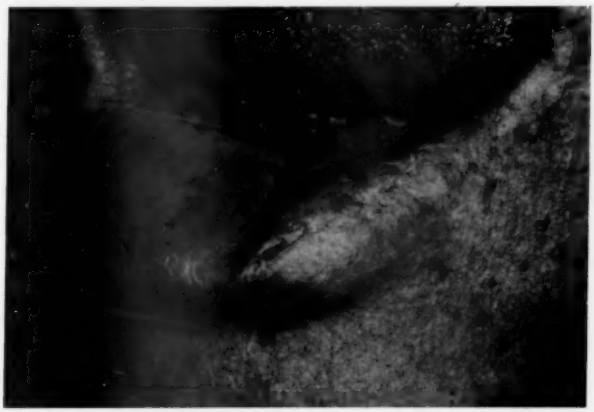

Fig. 2.—Glass slide over several lesions without application of pressure.

Fig. 3.—Lesions blanch from application of pressure, thus indicating that blood is intravascular.

On oral examination, small cherry-red areas were found on the lower lip, the dorsum of the tongue, the buccal mucosa, and the palate. Diascopy proved the spots to be of intravascular origin. The maxilla and the mandible were completely edentulous.

The complete upper denture which the patient had been wearing for three years had not caused the palatal lesions to hemorrhage. Likewise, the wearing of the new complete upper and lower prostheses constructed in our clinic has been uneventful.

DISCUSSION

The most significant point of this case is that, after many years of empirical treatment for a refractory anemia, the etiology of the systemic condition was established only when the patient's oral lesions were correctly diagnosed. In arriving at this diagnosis, the first step was to determine that the oral lesions were multiple areas of intravascularly contained blood. Hemangiomas and varices are other blood-containing lesions that had to be considered. The lesions in this patient were too small to be classified as hemangiomas. Varices, while most often seen on the ventral surface of the tongue, may occur in other areas of the mouth. They are rarely seen in such large numbers, however, and are larger, darker blue in color, and situated somewhat deeper beneath the overlying mucosa. The clinical appearance combined with the history permits the diagnosis.

It is particularly interesting that the patient described here was a Negro, since this is apparently only the fourth report in the literature of the disease occurring in members of the Negro race.

SUMMARY

A case of hereditary hemorrhagic telangiectasia in a Negro has been presented. In the medical literature there have been only three reports of the disease occurring in Negroes; no reports involving Negroes have appeared in the dental literature. In the case described here the etiology of severe hemorrhaging with resultant refractory anemia was not known for some forty years, until the observation of oral lesions led to the diagnosis.

A review of the literature is presented.

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EFFECT OF MASSIVE DOSAGE OF A NEW CORTICOSTEROID, TRIAMCINOLONE ACETONIDE, ON THE HARD AND SOFT TISSUES OF THE ORAL CAVITY IN THE NORMAL MONKEY

Edmund Applebaum, D.D.S., Austin H. Kutscher, D.D.S., Edward V. Zegarelli, D.D.S., M.S., and Henry P. Levy, D.D.S., New York, N. Y.

TRIAMCINOLONE ACETONIDE is a new synthetic corticosteroid (a derivative of triamcinolone) which has been found to be unusually active when applied topically; it is also a fully active corticosteroid when administered systemically. This compound has been found particularly useful in the treatment of dermatologic lesions,[1] from which sites systemic absorption is relatively limited, and also of oral lesions,[2] from which sites systemic absorption can be postulated to be of a higher magnitude.

On a weight basis, triamcinolone acetonide (Kenalog) is a potent corticosteroid with powerful pituitary-suppressant activity. The advantages of triamcinolone acetonide lie in its increased topical activity rather than in a peculiarly potent systemic action. Such medication as the patient ultimately swallows during the course of treatment of oral lesions is, however, available for systemic action.

In view of the scarcity of information as to the specific effects (and their severity) of systemically administered triamcinolone acetonide in man, our study was undertaken to ascertain the histologic findings in the hard and soft tissues of the oral cavity of normal monkeys following the systemic administration of large doses of this drug.

MATERIALS AND METHODS

The oral tissues available for histologic study were obtained from ten young adult rhesus monkeys (five males and five females ranging in weight from 120 to 133 pounds) which had been included in routine chronic toxicity* studies of triamcinolone acetonide.

The animals were divided into three groups. Group I (three animals) served as experimental controls and received no triamcinolone acetonide. They

From the Department of Anatomy of the College of Physicians and Surgeons, Columbia University, and the Division of Stomatology, School of Dental and Oral Surgery, Columbia University.

*The tissues studied in this experiment were obtained from animals which were the subjects of chronic toxicity studies at The Squibb Institute for Medical Research.

were killed at seven, forty-two, and sixty-four days,* respectively. Group II (three animals) received 1 to 2 mg. of triamcinolone acetonide per kilogram daily. They were killed at twenty-six, sixty-five, and sixty-five days,* respectively. Group III (four animals) received 3 to 6 mg. of triamcinolone acetonide per kilogram daily. These latter animals were killed at twenty-two, sixty-four, sixty-five, and sixty-six days,* respectively. In all other respects (diet, cages, etc.), the animals in all groups were cared for in similar fashion. The corticosteroid was administered by tube feeding; hence, its potential activity on oral tissues could be the result of systemic action only.

Tissues from all four animals in Group III (the highest dosage regimen) and all three animals in Group I were studied. Tissues from the remaining three animals (Group II, the lower dosage group) were available for study if warranted.

After the animals were killed, their heads were placed in 10 per cent formalin for at least two weeks. Specimens of the oral mucous membranes from the cheeks, tongues, and lips were obtained for histologic study. In addition, block sections of the mandible with the enclosed first molar tooth and attached gingivae were obtained. All soft-tissue specimens were stained with hematoxylin and eosin; the hard structures were decalcified prior to similar staining.

In order to minimize the effects of such variables as biopsy site, anatomic differences, sectional planes, etc., all tissues were cut and stained in the most exacting and identical fashion possible.

All tissues remained coded ("blind") throughout the period of handling, processing, and histologic study.

RESULTS

Histologic studies of the hard and soft tissues of the mouth and jaws did not reveal significant changes in the monkeys treated with triamcinolone acetonide (Group III) as compared with the control animals (Group I).

DISCUSSION

Although the absence of deleterious histologic changes in the oral hard and soft tissues of the treated monkeys was definitely noted, these findings cannot be construed as being entirely indicative of negative findings for the body as a whole; nor can these observations be interpreted as evidence that there must have been an absence of clinical findings elsewhere.

Extreme care must also be taken in the projection of these data (in monkeys) to the human species, since differences in terms of dose response in particular and individual tissue responses in general are often of great importance.

Nevertheless, it is obvious that, on a weight basis, the dosage of triamcinolone acetonide was indeed massive when compared to the usual dosage for human beings. For a 70 kilogram man, a comparable dose of triamcinolone

*Kenalog (triamcinolone acetonide, Squibb) was not administered to animals on Saturdays and Sundays during the experimental period.

acetonide would be (for Group III) in the neighborhood of 210 to 420 mg. per day, as compared with 1.0 mg. per day in anticipated normal topical oral usage in man.

SUMMARY

The oral mucous membranes, jaws, teeth, and surrounding soft tissues of a group of normal monkeys were studied histologically following the oral systemic administration for prolonged periods and at high dosage of a new corticosteroid, triamcinolone acetonide.

Comparison of the tissues obtained from triamcinolone acetonide-treated and control animals revealed no significant differences in any of the oral tissues examined.

The authors express their gratitude to Mrs. Tanya Genningsen for technical assistance in the preparation of these tissues for histologic study; to Mr. B. Thomas of The Squibb Institute for Medical Research, under whose direction the chronic toxicity studies of which these animals were a part were conducted; and Mrs. Aldonna Skislak and Mrs. Helene W. Kutscher for their assistance.

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630 WEST 168TH ST.

A NEW THERAPY FOR SEVERE HERPES SIMPLEX

Frank T. Scott, D.D.S., Jacksonville, Fla.

HERPES SIMPLEX requiring medical care is primarily treated by the physician. Frequently, however, cases of herpes simplex are seen in dental practice. These involve herpetic gingivostomatitis and sometimes herpes labialis which are seen as sequelae to oral surgery or in patients whose resistance has been lowered by dental infection. It is presumed that the virus which causes the disease is present in the body in a dormant form and becomes active after local trauma or infection.

In severe cases of herpes simplex the lesions may not be confined to the region of the lips but may spread to the adjacent areas and even appear inside the mouth. Usually the lesions are extremely painful. The pain often continues for several days or, in severe cases, for one to two weeks. Topical medications, although useful, are of limited value in reducing the pain and swelling; they do not shorten the course of the disease.

The efficacy of Protamide (Sherman Laboratories), a processed and denatured proteolytic enzyme, in the treatment of herpes zoster,[1, 2] varicella,[3] and inflammatory neuritis[4, 5] following a virus infection suggested its possible merit in treating cases of herpes simplex.

A brief study was conducted to determine the effectiveness of Protamide in a series of twelve cases of severe herpes simplex. One ampule of Protamide (1.3 ml.) was administered intramuscularly on two successive days. The pain was generally relieved within one or two hours following the initial injection of the drug, and the lesions began to dry up after the second injection.

Complete follow-up was maintained on patients included in the study. In addition to the clinical observations of the healing of the lesions, the twelve patients were questioned concerning their well-being. Ten patients expressed gratification at being relieved of the painful symptoms. No side effects were seen in patients treated with this drug.

Four interesting case histories follow.

CASE 1.—Patient N. V., a 30-year-old woman, had acute herpes labialis extending over most of the lower lip as a sequela to extensive oral surgery. She was in severe pain, and there was considerable swelling of the lip. In addition to the discomfort and pain, the patient was disturbed about the uncosmetic aspect of the lesions and swelling.

One daily injection of Protamide was administered for two days. The patient reported relief from pain within an hour following the initial injection. The swelling disappeared and the lesions started to dry up on the second day of treatment. Two ampules of the drug gave this patient complete relief.

CASE 2.—Patient F. S., a 43-year-old man, had one vesicle on the lower lip, accompanied by severe pain and discomfort. The pain subsided within an hour following one injection of Protamide. One additional injection of the drug was administered on the following day. Symptoms were completely relieved, and the vesicles dried up without further treatment. However, the patient suffered another attack a month later. This time the vesicle was on the upper lip. Three daily injections of Protamide afforded complete relief of pain, and the symptoms disappeared. A few weeks later the patient had a third attack of herpes simplex, and three daily injections of Protamide were administered. Pain was not relieved after the injections, as in the previous two attacks, and the lesions progressed through the normal course. This patient had a history of frequent recurrences of herpes simplex in the past. After the third attack and subsequent treatment with Protamide, there have been fewer recurrences during the past two years.

CASE 3.—Patient C. H., a 37-year-old woman, was seen with blebs on the left third of the upper lip, which was generally swollen and extremely painful. Two daily injections of Protamide were administered. The patient expressed relief of pain within two hours after the first injection. Examination on the third day revealed a clearing-up of the lesions. No swelling or scab was noticed.

CASE 4.—Patient J. M., a 43-year-old woman, had small lesions around and inside the left and right corners of the mouth. She was in extreme discomfort. The lesions were very painful, and the patient could hardly open her mouth to talk. One Protamide injection was administered intramuscularly. On examination two days later, the patient was found to have made satisfactory progress following the single injection. No further treatment was necessary.

DISCUSSION

These are typical examples of the response obtained with Protamide therapy in the cases of herpes simplex investigated in this study. In most cases excellent results were obtained within one to two hours following administration of the initial injection. Two injections were usually sufficient to afford complete relief from pain and swelling. The drug definitely seemed to retard the progress of the eruption and to reduce the time required for healing of the lesions in ten of the twelve cases studied.

In all cases an ointment containing 0.5 per cent of Neo-Cortef ointment was applied to the lesions, in conjunction with Protamide, to diminish local irritation. This topical ointment was found to be of value in relieving the pain and keeping the patient more comfortable, but it did not reduce swelling or shorten the duration of the lesions when used in the absence of Protamide injections.

SUMMARY

The therapeutic effectiveness of Protamide was investigated in a series of twelve cases of severe herpes simplex seen in dental practice. Injections (1.3 ml.) were administered intramuscularly on two successive days. Excellent results were obtained in most patients and no side effects were seen. It appears that if the drug is going to be effective, relief should be obtained following the first two injections. Protamide is sufficiently effective to warrant its use in patients with severe herpes simplex. In the twelve cases studied, ten patients responded favorably and two showed no response.

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ORAL ROENTGENOLOGY

American Academy of Oral Roentgenology

Arthur H. Wuehrmann, Editor

CURRENT STATUS OF THE POSTGRADUATE AND GRADUATE CURRICULA IN ORAL ROENTGENOLOGY

Arthur H. Wuehrmann, D.M.D., Birmingham, Ala.*

BEFORE I discuss the current status of postgraduate and graduate curricula in oral roentgenology, it seems appropriate to define terms which will be used throughout this article and which may have a varying connotation for the audience. In the *Report on Graduate and Postgraduate Programs in Dentistry, 1956-57,* the Council on Dental Education of the American Dental Association defined the word *program* as "a planned sequence of courses designed to provide the educational experience and training required for the acquisition of an advanced degree or a certificate of accomplishment." The Council specifically mentioned that a program does not have the same meaning as a course. *Graduate program* was defined as "a planned sequence of courses leading to an advanced degree such as an M.S., M.S.D., or Ph.D., granted by a recognized educational institution." *Postgraduate program* was classified as "a sequence of courses that does not lead to a degree, but one for which the student *may be* awarded a certificate. The level of instruction in the postgraduate program may or may not be similar to that in the graduate program, and the student may not be required to meet the same entrance requirements expected of the student registered for the graduate degree." The Council's report does not define *refresher program.* For purposes of this paper, refresher courses are those which vary in length from one day to approximately two weeks and which are designed primarily for the purpose of maintaining and advancing the knowledge of the professional person in areas closely related to the general or specialized practice of dentistry. In some instances, such courses are called "postgraduate." This is obviously the prerogative of the individual

Presented at a Workshop on the Teaching of Radiology in Dentistry, held March 18 and 19, 1960, at the College of Dentistry, University of Illinois, Chicago, Illinois.

*Associate Dean and Professor of Dentistry, School of Dentistry, University of Alabama; member, American Academy of Oral Roentgenology.

or school giving the course, but for purposes of clarification in this presentation, postgraduate programs, as defined above, are not synonymous with refresher courses.

In an effort to determine the activities of the American and Canadian dental schools in the area of postgraduate and gradute education in oral roentgenology, a letter was sent to the dean of each school with a request that he refer it to the staff member responsible for his department of oral roentgenology. The letter contained a very short questionnaire designed to obtain information which could be used as a base line for this discussion. In essence, the questionnaire asked the following:

1. Does your school offer:
   (a) refresher courses,
   (b) postgraduate courses of at least six months' duration leading to a certificate, and/or
   (c) graduate courses of at least one year's duration leading to a degree in the field of either roentgenology or radiology per se, or in a combined form with one or more clinical or basic areas of instruction?
2. If your school participates in a, b, and/or c of Question 1, what is the content of such a program?
3. If your school does not participate in a, b, and/or c of Question 1, do you feel that there is a need for and any immediate prospects of offering such instruction in your school?

Forty-eight replies were received from a total of fifty-one inquiries. The results are pertinent to this workshop.

Two or three schools (the reply of one school was ambiguous, hence the "two or three") indicated that they offer postgraduate programs, and five institutions stated that they offer graduate work. One of these combines graduate work in roentgenology with that in endodontics. Eleven schools offer what might be considered "service" programs in oral roentgenology for other graduate activities. It is entirely possible that this number may be low, since the question regarding the availability of such supporting courses was not raised in the questionnaire. Approximately 50 per cent of those replying did not comment on the need for graduate or postgraduate programs in oral roentgenology. Of the 50 per cent that did comment, most of them expressed the opinion that continuing education in this area is needed. Some of this group indicated that future courses were contemplated at their institution, but the larger proportion suggested that future courses in oral roentgenology were probably impossible because of a lack of staff and facilities. Many of the group that did not comment on need were included in those that are already presenting continuing education opportunities.

The scope of the refresher type of course varied, depending upon duration and on whether the program was designed for dentists or for auxiliary personnel. The programs for dentists ordinarily included both technical and

interpretive phases of the science. In some instances, the courses were restricted to either technique or interpretation or to the problems associated with radiologic health. Although approximately 50 per cent of our institutions do offer or have given refresher-type courses, the replies to the questionnaire suggest that the response to these programs is not as enthusiastic as might be hoped for and that the courses are given on the basis of demand rather than as an annual or semiannual event.

Postgraduate opportunities for study in oral roentgenology are offered at the University of Illinois, the University of Alabama, and possibly at Howard University. There was some question in the reply from Howard as to whether the postgraduate course described was of a refresher nature or was actually of approximately six months' or more duration. Since the postgraduate program at the University of Alabama is in many respects identical to the graduate program, the curriculum will be discussed under the graduate program. The University of Illinois is the only school offering a program specifically in the postgraduate area. It will be of interest to summarize this program briefly:

Objectives of the Course:

1. To provide a training program for dentists contemplating a teaching career in radiology.
2. To train dentists in preparation for eventual board certification.
3. To train research and teaching personnel in allied fields of dentistry.

Length of the Course:

The course is of one year's duration, requiring full-time attendance, and leads to a certificate in radiology upon successful completion of requirements.

Curriculum:

Radiology

1. Undergraduate lecture courses
   (a) Radiographic anatomy of the head (11 hours)
   (b) Radiology and oral diagnosis (11 hours)
   (c) Radiographic interpretation of pathologic changes (11 hours)
   (d) Applied radiographic interpretation and techniques (11 hours)
2. Clinical Practice
   (a) Plain extraoral radiography
   (b) Intraoral radiography
   (c) Special extraoral radiography
      (1) Laminagraphy
      (2) Rotography
      (3) Sialography
      (4) Cephalometry

3. Seminars
   (a) Research methods in radiology
   (b) Radiographic anatomy of the head
4. Review of the Literature

Electives

1. Histology
2. Oral pathology
3. Biochemistry
4. Oral surgery (hospital dental clinic)
5. Oral medicine

The questionnaire did not ask how long the graduate and/or postgraduate programs had been in existence or how many students were currently in residence. It did not request information about the number of persons who had satisfactorily completed the course work in past years.

The number of schools offering graduate work in roentgenology and radiology is not large. Opportunities are available at Washington University, the University of Pennsylvania, Indiana University, the University of Michigan, Tufts University, and the University of Alabama. The course content varies in each school, but in general considerable similarity exists. Curriculum in each of the schools includes most of the subjects listed in the postgraduate program at the University of Illinois plus additional activity in the basic sciences and in research. A thesis is generally required in any program leading to a master's degree. It may be beneficial to summarize the philosophy which exists at the University of Alabama School of Dentistry relative to postgraduate and graduate work in radiology, since I am well informed about these opportunities.

The graduate and postgraduate programs in oral roentgenology at the University of Alabama School of Dentistry are similar in many respects. The postgraduate program can be completed in one academic year; the graduate program requires a minimum of two academic years and the intervening summer. The graduate program is oriented toward research and study in the basic science areas to a greater extent than is the postgraduate activity. A thesis is expected for degree students. As in most other graduate programs, the thesis must be defended through a suitable oral examination. A specific outline of required and suggested elective courses is not available. The philosophy exists throughout the graduate programs at the University of Alabama School of Dentistry that courses leading to a degree should be planned on an individual basis with the enrollee. This should be done in accordance with his needs and objectives and then should be cleared with the dean of the Graduate School. It probably would be helpful to quote from correspondence mailed to persons who have inquired about graduate work in roentgenology at the University of Alabama:

> Fundamental to an understanding of oral roentgenology is basic information in oral medicine, oral diagnosis, and oral pathology. Basic to the latter is a good working knowledge of histology. In our graduate and postgraduate program, particularly the

former, we require that the student be thoroughly informed in these areas, and we make this type of learning experience available to him. Naturally, we expect the individual to participate in our undergradute roentgenology lectures, and this is supplemented by a postgraduate course in oral roentgenology which is given to many of our graduate and postgraduate students in other diciplines. In addition to this, clinical experience in roentgenographic techniques, both intraoral and extraoral, is given until the individual is certified as proficient in all aspects of technical work. The technique is supplemented by seminars in interpretation and correlated with research being done by the individual and that being actively carried on in the department. Within the period of one academic year, it is not practical for the postgraduate student to pursue his own research efforts, but he is made familiar with research methods and the research programs of others. The candidate for the graduate degree is expected to pursue individual original research.

It is interesting to note that a number of schools give supplementary or supporting courses for graduate students in other graduate and postgraduate programs. In formulating the questionnaire distributed to all the dental schools, no question was raised relative to such efforts. For this reason, the survey results are undoubtedly incomplete. It is possible to say with authority, however, that at least eleven schools include formal course work in roentgenology in the course content of other graduate and postgraduate activities. The course outlines used in these eleven schools are not available, but no doubt they would be similar to that offered at the University of Alabama. In the hope that such an outline will be helpful to others, it is included:

Physical principles of x-radiation	2 hours
Biologic effects and hazards of radiation	2 hours
Appraisal of recent advances in radiographic techniques and uses of ionizing radiation in medicine and dentistry	3 hours
Factors influencing film quality	3 hours
Extraoral roentgenographic techniques: These lectures are supplemented with demonstrations and clinical assignments. The student and his advisor are made aware that 20 hours will be required to complete the clinical work.	6 hours
Radiation therapy	2 hours
Osteology and roentgenographic anatomy of the skull and jaws	6 hours
Localization techniques and interpretation of caries	2 hours
Interpretation of apical pathology and periodontal disease	2 hours
Disease entities having predominantly roentgenolucent characteristics and disease entities having predominantly roentgenopaque characteristics	4 hours
Examination	

It is obvious, from the comments received from the various schools, that there is a need for further study of dental roentgenology by general practitioners and specialists. As is often the case, need and demand differ sharply. There is not a great demand within the dental profession for refresher-type courses or for graduate or postgraduate programs in oral roentgenology. At least some of the reasons for this are evident. The dentist, whether he is a general practitioner or a specialist, is his own *roentgenographic specialist.* Ordinarily, he alone judges his competency; often he is easily satisfied. He has not been trained to refer patients to roentgenologists. Indeed, if he did, there would be an insufficient number of roentgenologists to meet the demand. As a result of this lack of specialization, the potentials of the field have not been developed. Thus, a vicious cycle exists, and it is difficult to know exactly how this trend is to be changed.

A second reason for the lack of development in this field of ionizing radiation is the lack of time and effort which our teachers devote to teaching and research. This statement is not made in condemnation of teachers; rather, it emphasizes the present state of affairs which has developed largely because of inbreeding and economics. For years, dental school administrators have looked upon the department of roentgenology as a "service activity." They often have not seen it as an area that is truly essential to the intelligent practice of dentistry or as an area of study with almost unlimited research potentials. Far too many of our teachers devote several half-days to their academic activities and the remainder of their time to earning a living in either general practice or in the specialty of roentgenology. Perhaps those who do devote the major portion of their time to teaching or to the specialty practice of roentgenology are to be condemned somewhat more severely than the others for not having developed the field more extensively.

Oral roentgenology or radiology has by no means kept step with the manifold activities of medical radiology. Yet, it is a field which has at least as many potentials. This lack of new and interesting data can be exemplified by citing the dearth of publishable material currently being submitted to the editor of the American Academy of Oral Roentgenology for publication in ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY. Perhaps as much as 50 per cent of that submitted and published lacks depth as well as an understanding of true research methodology. This picture, of course, does have its bright side. There are some in the field now who are contributing significantly to its development, and it is hoped that their efforts will have a motivational effect on others in the relatively near future.

There is a serious question in my mind as to whether efforts should be made toward developing the area of oral roentgenology as a specialty. The more one deliberates, the more one is impressed with the need for specialization if reasonably rapid growth is to be obtained. The control of ionizing radiation in all its aspects is becoming more of a public health problem, and the roentgenographic practices used in medicine and dentistry will come under close scrutiny in the near future. If the general use of ionizing radiation in dentistry were

restricted to those who had established their competence, the science would undoubtedly grow with ever-increasing strides and the public would not only be more effectively protected from the harmful effects of ionizing radiation but would undoubtedly receive more adequate technical and diagnostic services.

Unfortunately, the complete withdrawal of ionizing radiation from the dental office would interfere materially with the quality of the technical service in other areas of dentistry. For example, the practice of endodontics would not be possible without the findings supplied by the x-ray film. Furthermore, the observance and the elimination of overhanging restorations and the location and removal of fractured root tips are dependent upon roentgenographic evaluation *at the time of need.* The x-ray machine is in the dentist's office to stay. The American Dental Association would strenuously resist any attempt to withdraw from the dentist his prerogative of using ionizing radiation, this is probably as it should be. Nevertheless, since the patient would benefit from the restricted use of ionizing radiation by the average practitioner, it may be advisable for those who have a major interest in oral roentgenology to take deliberate steps for the benefit of the patient and for the advancement of the science. Under these circumstances, there would be considerable demand for formal graduate work in our dental schools.

VARIATIONS IN X-RAY BARRIER EFFICIENCY OF SOME COMMONLY USED BUILDING MATERIALS

Walter S. Moos, Ph.D., Richard E. Haas, B.A.,* Seymour H. Yale, D.D.S.,** and J. Donald Hauptfuehrer,** Chicago, Ill.*

INTRODUCTION

CURRENTLY, the majority of dental offices in the United States are equipped with x-ray machines. The growing popularity of multiple-office professional buildings poses a special problem in the control of radiation emanating from x-ray machines in adjacent offices.

AIM OF THE PROJECT

The aim of this research project was to determine the relative x-ray barrier efficiency of some commonly used building materials in order to provide additional methods of reducing office-to-office radiation.

MATERIALS

X-ray Machines.—Two dental x-ray machines were used in this study: (1) Weber Model No. 11, operating at a nominal kilovoltage range of 45 to 70 KVP, with 0.375 mm. copper and 0.5 mm. aluminum added filtration[1] and (2) Weber Model 12 RM, operating at a nominal kilovoltage range of 40 to 90 KVP with 0.125 mm. copper and 0.5 mm. aluminum added filtration. Both machines were collimated to produce beam diameters of 2.75 inches at cone tip.

Barrier Materials.—With the exception of concrete, the barrier materials listed in Table I were prodcured on the open market. The concrete was prepared from stock material in the proportions of three parts gravel, two parts sand, and one part Portland cement. The mix was permitted to set for forty-eight hours. Each material was cut into slabs varying in surface size from 6 x 6 to 9 x 9 inches. The thickness of each material examined varied in accordance with the thickness commonly used.

Detection Instruments.— Two radiation-measuring devices were used: (1) Westinghouse Roentgen Probitron, Model FAX16X, and (2) Victoreen ionization chamber (1 r), Model 70.

*Department of Radiology, College of Medicine, University of Illinois.
**Department of Radiology, College of Dentistry, University of Illinois.

METHODS

1. The percentage of x-ray transmission was determined for each material in its commonly encountered thickness.

2. The beam intensities were measured with and without interposed absorbers by means of the Westinghouse Roentgen Probitron. This instrument was checked for x-ray energy dependency and accuracy against the Victoreen ionization chamber.

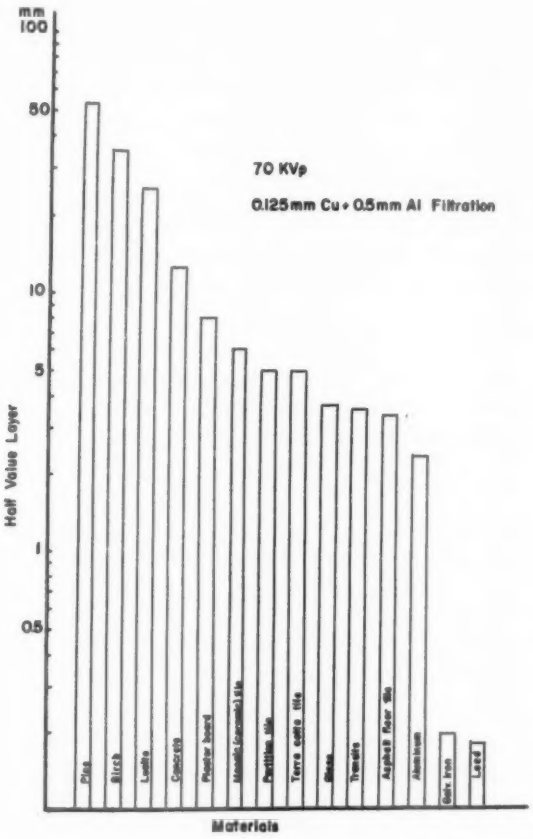


Fig. 1.

3. The target-to-instrument distance was 40 cm. with the absorber surface facing the tube located at 17 cm. from the target.

4. The absorption efficiency, described in terms of half-value layers,* was determined for most of the building materials, using six different beam qualities.

*Half-value layer is defined as that thickness of a material that will reduce beam intensity by one-half.

TABLE I. ABSORPTION VALUES FOR VARIOUS BUILDING MATERIALS AND DIFFERENT X-RAY ENERGIES

MATERIAL	COMMONLY ENCOUNTERED THICKNESS OF THIS MATERIAL IN INCHES	KV.: 70 PRIM. VOLT.: 110 FILTER: 0.125 MM. COPPER + 0.5 MM. ALUMINUM HALF-VALUE LAYER MM. ALUMINUM: 2.3		KV.: 60 PRIM. VOLT.: 110 FILTER: 0.125 MM. COPPER + 0.5 MM. ALUMINUM HALF-VALUE LAYER MM. ALUMINUM: 2.0		KV.: 50 PRIM. VOLT.: 110 FILTER: 0.125 MM. COPPER + 0.5 MM. ALUMINUM HALF-VALUE LAYER MM. ALUMINUM: 1.5	
		PER CENT TRANSMITTED FOR THIS COMMONLY ENCOUNTERED THICKNESS	HALF-VALUE LAYER OF MATERIAL (MM.)	PER CENT TRANSMITTED FOR THIS COMMONLY ENCOUNTERED THICKNESS	HALF-VALUE LAYER OF MATERIAL (MM.)	PER CENT TRANSMITTED FOR THIS COMMONLY ENCOUNTERED THICKNESS	HALF-VALUE LAYER OF MATERIAL (MM.)
Pine	9/16	80	53.0	80	50.0	78	45.0
Birch	9/16	74	35.0	72	33.0	70	30.0
Lucite	3/8	78	25.0	76	22.0	67	18.0
Plaster board (dry wall)	3/8	40	8.0	32	6.5	23	4.5
Mosaic tile (ceramic)	0.4	29	6.0	22	4.5	--	3.5
Partition tile (clay)	¾ (1½)	< 1	ca 5.0	< 1	4.0	< 1	--
Window glass	1/8	52	3.6	45	3.1	37	2.0
Transite	3/16	34	3.5	24	3.0	16	2.0
Asphalt floor tile	1/8	49	3.3	44	3.0	35	2.5
Galvanized iron	1/32	4	0.2	< 1	0.1	< 1	--
Cabinet steel (painted)	1/20	1.5	0.1	< 1	--	< 1	--
Terra-cotta tile	1⅛-1⅜	< 1	ca 5.0	< 1	--	< 1	--
Waylite tile	3	< 1	--	< 1	--	< 1	--
Press tile (brick)	2⅛	< 1	--	< 1	--	< 1	--
Concrete (3 parts gravel, 2 parts sand, 1 part cement)	1½	< 1	--	< 1	--	< 1	--

Conditions of Measurements:

Weber dental x-ray unit (U.I.), Model 11.
Stabilized voltage provided.

TABLE II. ABSORPTION VALUES FOR VARIOUS BUILDING MATERIALS AND DIFFERENT X-RAY ENERGIES

MATERIAL	COMMONLY ENCOUNTERED THICKNESS OF THIS MATERIAL IN INCHES	KV.: 90 PRIM. VOLT.: 110 FILTER: 0.375 MM. COPPER + 0.5 MM. ALUMINUM HALF-VALUE LAYER MM. ALUMINUM: 5.7		KV.: 75 PRIM. VOLT.: 110 FILTER: 0.375 MM. COPPER + 0.5 MM. ALUMINUM HALF-VALUE LAYER MM. ALUMINUM: 4.7		KV.: 60 PRIM. VOLT.: 117 FILTER: 0.375 MM. COPPER + 0.5 MM. ALUMINUM HALF-VALUE LAYER MM. ALUMINUM: 3.2	
		PER CENT TRANSMITTED FOR THIS COMMONLY ENCOUNTERED THICKNESS	HALF-VALUE LAYER OF MATERIAL (MM.)	PER CENT TRANSMITTED FOR THIS COMMONLY ENCOUNTERED THICKNESS	HALF-VALUE LAYER OF MATERIAL (MM.)	PER CENT TRANSMITTED FOR THIS COMMONLY ENCOUNTERED THICKNESS	HALF-VALUE LAYER OF MATERIAL (MM.)
Pine	9/16	88	76.0	86	75	80	60.0
Birch	9/16	82	55.0	81	52	--	50.0
Lucite	3/8	--	--	--	--	--	--
Plaster board (dry wall)	3/8	62	16.0	60	12.7	58	12.0
Mosaic tile (ceramic)	0.4	52	10.0	46	9.0	--	--
Partition tile (clay)	¾ (1½)	26	9.3	21	8.4	--	--
Window glass	1/8	74	7.2	69	5.9	--	--
Transite	3/16	59	6.3	54	5.4	52	5.1
Asphalt floor tile	1/8	73	7.2	68	5.9	--	--
Galvanized iron	1/32	23	0.4	15	0.3	11	0.3
Cabinet steel (painted)	1/20	14	0.4	6	0.3	--	--
Terra-cotta tile	1⅛-1⅜	--	--	--	--	--	--
Waylite tile	3	--	--	--	--	--	--
Press tile (brick)	2⅛	--	--	--	--	--	--
Concrete (3 parts gravel, 2 parts sand, 1 part cement)	1½	--	--	--	--	--	--

Conditions of Measurements:

Weber dental x-ray unit (U.I.), Model 12 RM.
Stabilized voltage provided.

FINDINGS

1. For purposes of comparison, absorption characteristics of various building materials expressed in half-value layers are shown in Fig. 1.

2. Galvanized iron is an effective barrier material for x-ray energies commonly encountered in dental offices, comparing relatively well with the absorption qualities of lead.

3. Materials such as wood, dry wall, etc., frequently used for wall partitioning, are poor absorbers of x-radiation.

4. A complete summary of all findings is shown in Tables I and II.

SUMMARY

A study was made of the variations in x-ray barrier efficiency of some commonly used building materials. The investigation was initiated because the growing popularity of multiple-office professional buildings presents a special problem in the control of radiation emanating from x-ray machines in adjacent offices.

REFERENCE

1. Yale, S. H., and Goodman, L. S.: Reduction of Radiation Output of the Standard Dental X-ray Machine Utilizing Copper for External Filtration, J. Am. Dent. A. 54: 354, 1957.

ROENTGENO-QUESTIONS

Patients often ask, "How often should my teeth be x-rayed?" How can this question be answered intelligently, especially in light of the current concern over radiation hazards?

The average patient should have a full-mouth dental x-ray examination at least every five years. This will vary according to the patient's dental health. In addition, single films are recommended for any one complaint in any particular area, and bitewing films should be taken about once a year.

These examinations should keep the patient within safe dosage limits if the dental x-ray unit is safe and if modern techniques with fast film are used. The dental unit should be monitored by a qualified person, regardless of the age of the unit, and the equivalent of approximately 2 mm. total aluminum filtration and a skin surface exposure area 2¾ inches in diameter should be used.

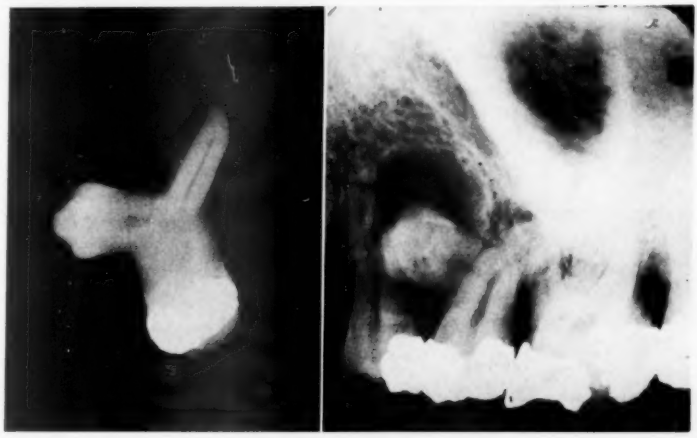
The above statements refer to routine x-ray examinations for the average patient. The individual case must be evaluated separately; for example, a person with rampant caries may need routine bitewing roentgenograms every six months. An evaluation of the need for the particular x-ray examination and the probable hazard of the examination should be made by the dentist before any roentgenogram is made.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

Root Fusion

THE roentgenograms reveal what appears to be a supernumerary tooth partially hidden by a maxillary second premolar, with the former encapsulated by a dentigerous cyst. Although there were no specific symptoms of a strictly dental nature, the patient had sought care because of a Bell's palsy on this side.



Surgical removal demonstrated fusion of a supernumerary crown to the root of the second premolar. The roentgenographic evidence suggests a common pulp chamber.

Benjamin S. Recant and Paul S. Kaufman
Long Island City, N. Y.

Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

ORAL PATHOLOGY

General Section

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TUBERCULOSIS OF THE SALIVARY GLANDS

A Collective Review

William B. Donohue, D.D.S., Montreal, Quebec, and*
*Theodore E. Bolden, D.D.S., Ph.D.,** Jersey City, N. J.*

INTRODUCTION

SWELLINGS in the region of the salivary glands pose a problem in differential diagnosis. Among the entities which may be seen occasionally are tubercular swellings of the salivary glands. Most reports of oral tuberculosis have dealt with lesions of the tongue,[1-3] the jaws,[4-8] gingiva,[9] lymph nodes,[10] nasal bones,[11] and occasionally the lips. Tuberculosis of the salivary glands has been reported sporadically and infrequently.[12-15] Because of the paucity of cases in the literature and the lack of information in the standard textbooks, this review of the subject is submitted and a new series of cases is reported.

REVIEW OF THE LITERATURE

Incidence.—De Paoli[15] reported the first case of tuberculosis of the salivary glands in 1893. Since then some seventy-nine additional cases have been recorded.[16-59] From this collection, it is evident that the parotid glands are more frequently involved by tuberculosis than any of the other salivary glands. The submaxillary gland is next in order of frequency of involvement, while only three cases of a tubercular swelling of the sublingual gland have been cited (Table I).

This investigation was supported in part by a research grant, B-1722, from the Department of Health, Education, and Welfare, United States Public Health Service, Council on Neurological Disease and Blindness.

*Department of Pathology, Faculty of Dental Surgery, University of Montreal, and the Departments of Dentistry, Division of Oral Surgery of the St. Mary's Memorial, Queen Elizabeth, Reddy Memorial, and Notre-Dame Hospitals, Montreal, Quebec. Formerly, Department of Oral Surgery, Cook County Hospital, Chicago, Illinois.

**Department of Oral Diagnosis and Pathology, Seton Hall College of Medicine and Dentistry, Medical Center, Jersey City, New Jersey.

About one-third of the cases occurred during the second and third decades of life. There was an almost equal incidence in male and female patients. Glandular swellings were present from three or four days up to thirty-five years before a diagnosis of tuberculosis was made.

TABLE I. SUMMARY OF DATA ABSTRACTED FROM SEVENTY-NINE CASES OF TUBERCULOSIS OF THE SALIVARY GLANDS COLLECTED FROM THE LITERATURE

AGE AND SEX INCIDENCE		LOCATION		DURATION OF LESION BEFORE A DIAGNOSIS WAS ESTABLISHED	PROVISIONAL DIAGNOSIS, BEFORE DIAGNOSIS OF TUBERCULOSIS WAS ESTABLISHED	
AGE (YEARS)	NO. OF CASES	GLAND INVOLVED	NO. OF CASES	GLAND AND SEX	PROVISIONAL DIAGNOSIS	NO. OF CASES
1-10	6	Parotid	57	Parotid	Neoplasm	24
11-20	9	Right	20	Males—3 or 4 days to 10 years	Malignant	5
21-30	13	Left	19		Benign	19
31-40	7	Bilateral	4	Females—20 days to 35 years	Inflammation	6
41-50	11	Not indicated	14		Abscess	3
51-60	8				Chronic	2
61-70	5	Submaxillary	19	Submaxillary	Cyst	1
71-80	1	Right	5	Males—2 months to 6 or 7 years	Infection	10
Not indicated	19	Left	1		Actinomycosis	1
		Bilateral	2	Females—4 months	Mumps	1
SEX INCIDENCE		Not indicated	11		Nonspecific	1
Males	29				Tuberculosis	7
Females	30	Sublingual	3	Sublingual	Obstruction	0
Not indicated	20	Not indicated	3	Not indicated	Not indicated	39

Preoperative Diagnosis and Systemic Findings.—It is interesting to note that only seven of the seventy-nine cases were correctly diagnosed, preoperatively, as tuberculosis (Table I). The most frequent diagnosis was benign mixed tumor. Syphilis, actinomycosis, acute and chronic inflammation, and malignant neoplasm were also considered. In three instances draining fistulas were associated with the swelling. No family or personal history of tuberculosis was elicited in approximately 75 per cent of the cases. In these, the tubercular infection was presumed to be primary to the salivary gland.

Scudder[56] described a typical case of primary tuberculosis of the right parotid gland in a 57-year-old woman. The lesion was first observed five years previously as a small swelling just below the lobule of the right ear. It had slowly increased in size but presented no subjective symptoms.

Secondary involvement of the salivary glands in a generalized tubercular process is quite variable. Pinoy,[60] after having systematically checked the salivary glands of twenty-six patients with proved tuberculosis at autopsy, could affirm that he had never detected any glandular lesion.

Kirch,[38] on the other hand, recovered salivary glands from patients who had died of miliary tuberculosis. He found tubercular lesions in six of thirteen submaxillary glands, one of nine parotid glands, and two of six sublingual glands examined histologically.

Gross Features.—Clinically, there appear to be two distinct types of salivary tubercular lesions. One is a chronic or encapsulated type which may

not produce symptoms for many years and is usually associated with a circumscribed swelling. The second is an acute inflammatory lesion which is diffuse and runs its course in a matter of days or weeks. It is usually associated with a generalized glandular swelling. In six of the recorded cases pus or purulent material could be expressed from the involved gland or its duct.[18, 25, 42]

The swellings may be hard,[15, 43, 44] soft,[20] or fluctuant.[19, 42] They are usually described as *small* tumors, but they may have the volume of a large almond or a small pigeon egg,[19, 52] a hazelnut,[50] or a walnut.[19]

Pain is usually a late manifestation.[40, 58] Partial facial paralysis has been a rare associated finding.[15, 26] Generally, the lesions are freely movable, but they may be attached to the skin.[14, 40]

Histologic and Bacteriologic Features.—In fifty cases the final diagnosis of tubercular salivary gland or tuberculosis of the salivary gland was made on the basis of histologic analysis. Two diagnoses were made on the basis of bacteriologic study.

The histologic features in the salivary glands were not unlike those of tuberculosis found in other tissues. They included (1) inflammatory changes and caseation necrosis,[45] (2) various foci of epithelioid, small round, and numerous giant cells,[56] (3) loss and destruction of parenchyma,[15, 47] and (4) demonstration of tubercle bacilli within the excised tissue.[39] In three cases the tubercular infection was found to be associated with an adenolymphoma of the same gland.[24]

Guinea Pig Inoculation.—The inoculation of guinea pigs with materials recovered from the affected gland, without additional surgical intervention, was employed as a diagnostic tool.

Five cases were deemed to be tuberculosis on the basis of a positive tubercular reaction in the respective guinea pigs, four to six weeks following inoculation.[17, 32, 33]

Method of Spread.—Wood[59] emphasized the possibility of extension of tuberculosis from the tonsils to the parotid gland via the lymphatics. In the case that he reported both the parotid gland and the tonsils showed tubercular involvement. Wood pointed out that tuberculosis of the tonsils is common. It is possible that this method of spread may account for the greater occurrence of tuberculosis in the parotid gland.

It is also possible that tubercular infections around the teeth may extend to the salivary glands by the same pathway. Mendel,[65] however, was unable to produce a tubercular infection of the salivary glands via the dental route.

De Paoli[15] observed tubercular follicles along the pathway of the facial nerve. Mondor and Gauthiers-Villars[46] cite a case reported by Lemaitre which involved the parotid gland and which, he believed, traveled by way of the facial nerve trunk.

The more probable pathways of spread of tubercular infections to the salivary glands are by way of the hematogenous, lymphatic, and canalicular

routes. In most cases, the lymphatic and canalicular routes would seem to be incriminated, since in a hematogenous spread one would expect a more generalized miliary involvement.

Therapy.—Feuz[33] felt that tuberculosis of the salivary glands simulated an adenopathy and that conservative therapy should be employed. To date, therapy has been primarily surgical.[15, 35, 36, 39, 44, 45, 50] This reflects the rarity of the condition and the difficulty of establishing a precise diagnosis in swellings of the salivary glands without a surgical exploration and microscopic examination. The exception to this may be in glands secondarily involved in a generalized tubercular infection. Aspiration biopsy[49] and the inoculation of collected material into guinea pigs might help to establish the diagnosis.

In a few cases, ultraviolet rays,[17, 53] infrared light,[33, 60] and the injection of antiseptic oil into the excretory duct[28] have been employed with questionable regression of the lesion. Similar regression has been noted following the use of ultraviolet rays to treat tubercular fistulas involving the lacrimal gland.

The use of x-rays and local currettage of the lesion, followed by the application of zinc chloride[19] and tincture of iodine, has been suggested. Recently chemotherapeutic agents have been employed in the treatment of systemic or pulmonary tuberculosis.[3] Their promise for primary and secondary tubercular lesions in the oral cavity is great.

Experimental Tubercular Adenitis.—Pinoy[60] attempted to induce tuberculosis of the salivary glands experimentally in dogs by injecting viable bacilli into the duct of the submaxillary glands. Fiorani[61] and Franchetti and Menini[62] repeated Pinoy's work and were unable to produce tuberculosis except in a few instances where the gland was traumatized and the excretory duct was ligated following the canalicular injection.

In Pinoy's experiments, injection of tubercle bacilli into the carotid artery resulted in tuberculosis of the submaxillary gland at the end of one month. Similar results were achieved by the direct injection of bacilli into the gland.

Valude[63] speculated that the rarity of tubercular involvement of the salivary glands might be due to an antagonism to the tubercle bacillus from the numerous organisms of the oral cavity. Lucchese,[64] with this theory in mind, injected a solution composed of viable tubercle bacilli and saliva into the peritoneal cavities of guinea pigs. His control solution was composed of saline and tubercle bacilli. All animals developed tuberculosis. Lucchese postulated that the rarity of the disease in the salivary glands might be due to some basic characteristic of gland metabolism.

Carmody[21] suggested that this rarity might be due to the protection afforded by the stratified squamous epithelium. "We know that epithelium is not affected by tuberculosis, but whether the bacillus is rendered inert, for the time being, by passage through these cells and must lodge in a lymph gland to regain its virulence is a question."

Mikulicz's Syndrome, Mikulicz's Disease, and Tuberculosis.—Bilateral swelling of the salivary *and* lacrymal glands unattended by pain or systemic

changes was first described by Mikulicz and is known as Mikulicz's disease. Mikulicz's syndrome differs from Mikulicz's disease in one particular, namely, it presents a clinical picture as part of some other general disease, such as tuberculosis.

Three cases of Mikulicz's syndrome of tubercular origin have been authenticated by the demonstration of the tubercle bacillus in the excised tissue.[66]

Uveoparotid Tuberculosis.—Garland and Thompson[67] pointed out that, of the various agents thought to be responsible for inflammatory lesions of the uveal tract and of the parotid glands, tuberculosis is most often the sole etiological agent. For this reason, they suggested the name "Uveoparotid tuberculosis."

The clinical features of this syndrome are similar to those of uveoparotid fever and include bilateral enlargement of the parotid glands and chronic bilateral uveitis. The parotid glands never show suppuration, however, and the swelling usually subsides within months.

Two cases, one in which bilateral biopsies of the parotids had been performed, were offered in support of this position.[67] A third case of uveoparotid tuberculosis[68] was reported in a 17-year-old girl who presented with bilateral iridocyclitis, bilateral enlargement of the parotid glands, and a positive Mantoux reaction. Biopsy of the right parotid gland showed tuberculous systems with destruction of the gland parenchyma.

Some authors feel that uveoparotid tuberculosis and uveoparotid fever are actually the same disease.[69] Clinically, many cases of uveoparotid fever give no other suggestive evidence of tuberculosis, but at autopsy miliary tubercular lesions can be demonstrated.[69] Tait[70] has taken exception to the suggested role of *Mycobacterium tuberculosis* in the production of uveoparotitis.

CASE REPORTS

The five cases reported in this article were seen at the Cook County Hospital in Chicago, Illinois, between 1937 and 1957. Only those cases in which a histologic diagnosis was made are included in this series.

CASE 1.—Patient L. B., an 18-year-old Negro girl, reported to the oral surgery clinic, with the chief complaint of swelling in the left submaxillary region. The swelling had been present for about three weeks. There was no pain or discomfort.

Clinical Examination.—A freely movable mass was revealed in the area of the submaxillary gland.

Laboratory Data.—Chest x-ray examination was negative. Sputum was positive for T.B.C. Blood was negative for T.B.C.

Clinical diagnosis: Pleomorphic tumor of the submaxillary gland.

Hospital Course.—The patient was taken to the operating room where, under local anesthesia, the submaxillary mass was removed.

Pathology Report.—The specimen was fixed in formalin. It measured 2.1 by 4.0 by 2.0 cm. and was covered by a thin connective-tissue capsule. The cut surface was nodular, and the nodules were pale yellow.

Microscopic examination showed lobules of submaxillary gland separated by thick bundles of collagenous fibers. The lobules consisted predominantly of duct tissue; no

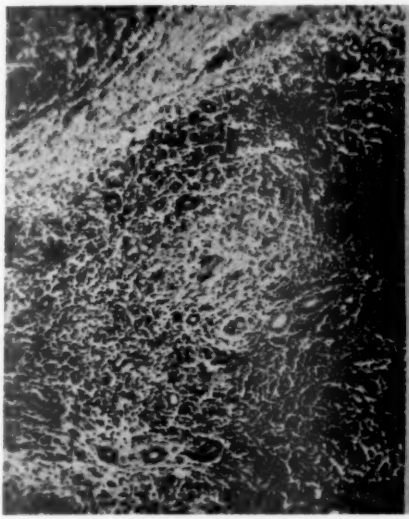

Fig. 1.

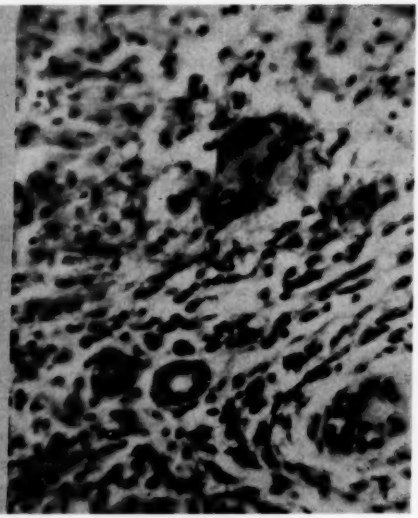

Fig. 2.

Fig. 1.—Case 1. Photomicrograph showing marked distortion of the lobule of a submaxillary gland. Few acini are present. The ducts are dilated, and the interstitial tissue shows marked infiltration by lymphocytes. A nodular area of caseation necrosis is observed in the center of the field. (Magnification, ×100; reduced ⅛.)

Fig. 2.—Case 1. Photomicrograph of nodular area of caseation necrosis shown in Fig. 1. Note the ductal elements, epithelioid cells, and lymphocytes. A giant cell of the Langhans type is seen at the top of the field. (Magnification, ×430; reduced ⅛.)

acinar structure was seen. The intralobular connective tissue was infiltrated by focal collections of lymphocytes. These surrounded nodular areas of caseation necrosis, epithelioid cells, and Langhans' giant cells (Figs. 1 and 2).

Diagnosis: Tuberculous adenitis of the submaxillary gland.

Fig. 3.

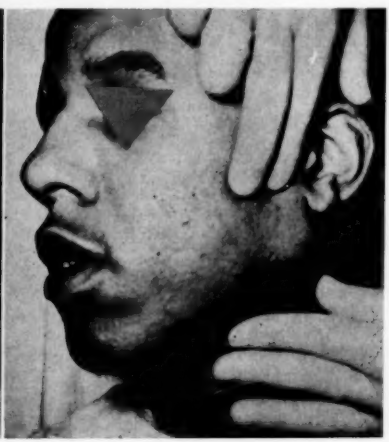

Fig. 4.

Figs. 3 and 4.—Case 2. Photographs showing firm mass in the area of the angle of the right mandible of a 21-year-old male patient.

CASE 2.—Patient H. R., a 21-year-old Negro man, reported to the oral surgery clinic with a swelling of the right side of the face.

Clinical Examination.—A firm, slightly tender, nonmobile mass was found in the area of the angle of the mandible on the right side. This mass measured approximately 4 by 3 cm. (Figs. 3 and 4). Small discrete lymph nodes were palpable along the right cervical chain. No purulent material could be milked from Stensen's duct. The swelling had been present for the past seventeen years and had been first noticed when the patient was 4 years old. There was no family history of tuberculosis.

Fig. 5.—Case 2. Photograph of extirpated mass from parotid region of a 21-year-old male patient.

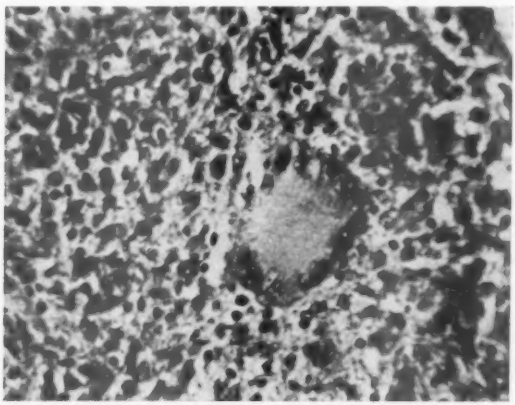

Fig. 6.—Case 2. Photomicrograph through section of extirpated mass from parotid region. Note the epithelioid cells, lymphocytic infiltrate, and the Langhans giant cell. (Magnification, ×430; reduced ⅙.)

Laboratory Data.—A chest film showed clear lung fields, except for a nodular calcification in the right midlung field. Urine was negative for albumin and acetone. Serology was negative. The Mantoux reaction was 1 plus with 1: 1,000 Old Tuberculin. The remaining laboratory findings were within normal limits.

Clinical diagnosis: Pleomorphic tumor of the parotid gland.

Hospital Course.—The gland was removed under general anesthesia and submitted for histologic examination.

Pathology Report.—The specimen was fixed in formalin. It measured 25 by 12 by 12 cm. (Fig. 5). The surface was smooth. The cut surface appeared grayish with white streaks.

Microscopically, the mass from the parotid gland showed, in one area, a fibrous capsule with slight lymphocytic infiltration. The bulk of the tissue consisted of larger and smaller foci of central necrosis, surrounded by epithelioid cells and a large number of lymphocytes (Fig. 6). Numerous giant cells of the Langhans type could be seen. In some areas accumulations of eosinophils were present. Occasionally remnants of ductal elements could be seen throughout the specimen.

Diagnois: Tuberculous adenitis of the parotid gland.

CASE 3.—Patient A. J., a 2½-year-old Negro boy, was brought to the ear, nose, and throat Clinic because of a swelling of the left side of the face.

Clinical Examination.—A preauricular swelling about 2.5 cm. in diameter, was found on the left side of the face. It was of rubbery consistency, nontender, and slightly mobile. Nothing could be expressed from Stensen's duct. The swelling had been present for three months and was slowly increasing in size. The patient had had no other illnesses, and there was no family history of tuberculosis.

Laboroatory Data.—Urine was negative for albumin and acetone.

Clinical diagnosis: Pleomorphic tumor.

Pathology Report.—A biopsy of the tumor led to the diagnosis of Boeck's sarcoid or hyperplastic tuberculosis of the parotid gland.

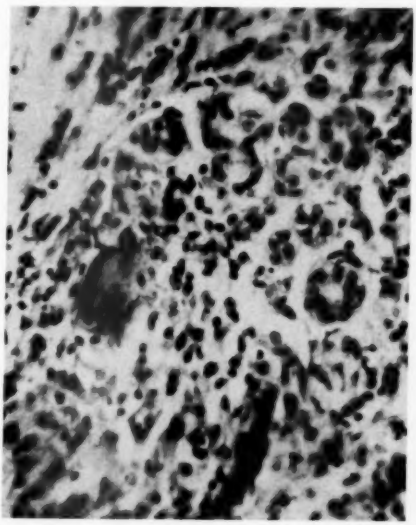

Fig. 7.—Case 5. Photomicrograph through lobule of the parotid gland. Note the ductal elements, epithelioid cells, lymphocytes, and the Langhans giant cell. (Magnification, ×430; reduced ⅛.)

CASE 4.—Patient F. K. was a 36-year-old Caucasian man who reported to the oral surgery clinic with a swelling of the right side of the face.

Clinical Examination.—A moderately firm, freely movable swelling of the right parotid area near the lobe of the right ear was revealed. The swelling had been present for several years. The patient was an epileptic, but he had no personal history of tuberculosis.

TABLE II. SUMMARY OF PERTINENT DATA IN FIVE CASES OF TUBERCULOSIS OF MAJOR SALIVARY GLANDS SEEN AT COOK COUNTY HOSPITAL, CHICAGO, ILLINOIS, 1937-1957

CASE NO.	AGE (YEARS)	SEX	RACE	DURATION OF LESION	PRESENTING SYMPTOMS	SYSTEMIC INVOLVEMENT	PROVISIONAL CLINICAL DIAGNOSIS	PATHOLOGY REPORT
1	18	F	Negro	3 weeks	Swelling on left submaxillary region; freely movable mass	Negative chest, blood; sputum positive for T.B.C.	Pleomorphic tumor of submaxillary gland	Tuberculous adenitis of submaxillary gland
2	21	M	Negro	17 years	Swelling of right side of face; firm nonmobile mass in area of angle of mandible	Negative for T.B.C.	Pleomorphic tumor of parotid gland	Tuberculous adenitis of parotid gland
3	2½	M	Negro	3 months	Periauricular swelling of left side of face	Negative for T.B.C.	Pleomorphic tumor	Boeck's sarcoid or hyperplastic tuberculosis of parotid gland
4	36	M	White	Several years	Swelling of right parotid area near lobe of right ear, freely movable	Negative for T.B.C.; epileptic	Benign tumor	Tuberculosis
5	28	M	Negro	2 months	Swelling of left side of face; mass near lobe of left ear, freely movable, semifluctuant; nodes in right cheek draining purulent material	Pulmonary TB, left lung	Tumor ? Cyst ?	Tuberculosis of parotid gland

Laboratory Data.—Serology was negative. There was a white blood count of 12,650, with a hemoglobin level of 87 per cent. The differential blood count revealed neutrophils 81 per cent, eosinphils 1 per cent, lymphocytes 7 per cent, monocytes 11 per cent, and microcytosis 1 plus.

Clinical diagnosis: Benign salivary tumor.

The pathology report indicated tuberculosis of a salivary gland.

CASE 5.—Patient W. R. was a 28-year-old Negro man who complained of a swelling of the left side of the face.

Clinical Examination.—A small mass located near the lobe of the left ear was revealed. The mass was freely movable and semifluctuant; it had been present for about two months and had been slowly increasing in size. Two small nodules were also noted in the right cheek near the commissure of the mouth. These drained purulent material and had been present for about one month. The patient had a history of pulmonary tuberculosis involving the left lung.

Laboratory Data.—A chest x-ray showed an area of radiolucency overlying the second anterior interspace. This appeared to be a cavity within the collapsed lung. A 50 per cent pneumothorax of the left chest was present. Urine was negative for albumin and acetone. Serology was negative.

Clinical diagnosis: Benign salivary tumor with necrotic degeneration.

Pathology Report.—The specimen was composed of salivary gland tissue which showed slight infiltration with lymphocytes. The connective tissue showed dense infiltration by round cells, especially lymphocytes, mononuclear cells, and occasional giant cells (Fig. 7).

Diagnosis: Tuberculosis of the parotid gland.

COMMENT

The five cases of salivary tuberculosis reported here were observed in four male patients and one female patient (Table II). The patients' ages ranged from 2½ years to 36 years, and the presenting symptoms (swelling of the face) had been present for periods ranging from three weeks to several years.

Distribution of these salivary lesions was in agreement with the data accumulated from the literature (Table I); 80 per cent of the cases involved the parotid gland, and the remaining 20 per cent involved the submaxillary gland. Involvement of the sublingual gland was not demonstrated.

Four of the five cases were primary. Tests failed to demonstrate any other foci of infection. One case was associated with a demonstrable tubercular lesion in the left lung.

Salivary gland tumor, benign or pleomorphic, was the provisional clinical diagnosis in every case. Tubercular involvement was not recorded, even in the one case of known pulmonary tuberculosis.

SUMMARY

1. The literature on tuberculosis of salivary glands has been reviewed and five new cases have been reported.

2. The major salivary glands may be the site of primary and/or secondary tubercular involvement. The sublingual gland is rarely involved; the parotid gland is the most frequent site.

3. Tuberculosis of the salivary glands presents a considerable problem in diagnosis. A correct preoperative diagnosis of salivary tuberculosis was not made in the majority of the cases presented, even in the presence of a history of pulmonary tuberculosis. Since this is an uncommon disease, it is probable that surgical exploration and the partial or total removal of the affected gland will remain in use until a more conservative diagnostic procedure can be established.

4. The clinical course of the disease in the salivary glands, whether primary or secondary, is unspectacular. In most instances there is no purulent exudate, either from the gland or from the duct of the involved gland. It is suggested that aspiration of the gland and guinea pig inoculations, along with chest x-rays, sputum analysis, and blood studies, may prove of value in establishing an early diagnosis of tuberculosis of the salivary glands.

5. It is quite possible that the incidence of tubercular involvement of the salivary glands is higher than is indicated by the few cases recorded in the literature. Many secondary tubercular salivary gland swellings may have been resolved with definitive therapy for and regression of the primary systemic lesion. It would be interesting to know the incidence of salivary gland swellings in patients with known systemic or pulmonary tuberculosis.

6. Tubercular involvement of the salivary glands in Mikulicz's disease, Mikulicz's syndrome, and uveoparotid fever would appear to be authentic.

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METASTATIC CARCINOMA OF THE MANDIBLE

R. H. Sage, F.R.C.S., and G. S. Hoggins, B.D.S., F.D.S.R.C.S.,
Birmingham, England

INTRODUCTION

THE tendency for certain tumors to metastasize in bone is well known. The common primary sites are the thyroid gland, breast, prostate gland, lungs, and kidneys.

In general, these bony metastases occur in the parts of the skeleton in which vascularity of the bone marrow persists into adult life, namely, the spine, ribs, sternum, pelvis, and proximal ends of the long bones. Metastases to the vault of the skull also occur sporadically.

According to Cohen,[1] bone metastases arise as a result of hemic spread, for the existence of lymphatic vessels in bone has never been demonstrated beyond dispute.

If bone vascularity is the predisposing factor in osseous metastasis, the lower jaw could not be considered a particularly likely site. Red marrow was found in only 25 per cent of a large number of mandibles examined, and then only in small discrete patches.[2] However, a survey of the literature over the last thirty years suggests that in clinical practice secondary mandibular neoplasms from the recognized primary sites are by no means rare. Osteolytic or osteoplastic secondary tumors may occur in the jaw, depending on the type of primary neoplasm.

Should any complaint of pain, swelling, or paresthesia in the mandible arise, without obvious dental cause, then the existence of a metastatic lesion in the jaw should be strongly suspected.

Not only the five main sources of primary tumors but also the less common sites of origin, such as the gastrointestinal tract, should be regarded as potential sites for spread to the mandible, as evidenced by the following case in which clinical and x-ray examination revealed an advanced osteolytic secondary tumor in the mandible following excision of an adenocarcinoma of the ascending colon.

CASE REPORT

A 62-year-old male patient, a cellarman by trade, first came to the outpatient department on Oct. 20, 1955. For six months he had suffered from epigastric pain, which came

From Selly Oak Hospital, Birmingham 29, England.

on half an hour after meals. There was no vomiting, but the patient suffered considerable flatulence. He was afraid to eat, since food made the pain worse. He admitted to no bowel symptoms whatsoever.

On examination, the patient was pale and had obviously lost weight. Abdominal examination revealed a hard mass, measuring approximately 4 by 3 inches, in the right upper quadrant of the abdomen. The mass was mobile and slightly tender. There was no clinical evidence of liver enlargement, free fluid, or lymph-gland enlargement.

Because of the pain's marked relationship to food, a barium meal was ordered on the assumption that the mass was a neoplasm of the pyloric end of the stomach. The barium-meal roentgenograms revealed a smooth narrowing of the pyloric antrum, more indicative of pressure from an extragastric mass than of an intrinsic gastric neoplasm.

Fig. 1.—Preauricular swelling.

In view of these radiologic findings, a barium enema was given. The roentgenograms showed a complete obstruction just below the level of the hepatic flexure of the ascending colon, where an annular filling constriction almost completely occluded the bowel.

Exploratory laparotomy was advised, and the patient was admitted to the hospital for this purpose on Nov. 15, 1955. The preoperative hemoglobin estimation was 12 Gm. per cent; urinalysis revealed no abnormality. After preliminary treatment with intestinal antiseptics and bowel washouts, the patient was given a general anesthetic and a laparotomy was performed through a right paramedian incision. A large mobile growth was found, involving 3 inches of the ascending colon and situated 3 inches below the hepatic flexure. There was no evidence of local peritoneal spread, and no metastases were found in the liver. A radical right hemicolectomy was performed; at least a foot of terminal ileum and the entire right half of the colon to the middle of the transverse colon were excised.

The patient stood the operation quite well and made an uneventful recovery. He was discharged twenty-one days after the operation. Microscopic examination of the surgical specimen proved the tumor was an adenocarcinoma of the colon with some malignant penetration of the muscles. One lymph gland was found to be involved.

The patient was seen in the outpatient department in January, 1956, when he looked well and had no complaints. On examination, his abdomen appeared quite satisfactory. He was seen again on Feb. 28, 1956, by which time he had gained 11 pounds.

Because he complained of precordial pain in May, 1956, the patient was referred to a physician. An electrocardiogram showed a normal rhythm but severe left ventricular strain. A Wassermann reaction and a Kahn test were negative. The patient was advised to take small doses of digitalis and to carry glyceryl trinitrate tablets to take if he experienced precordial pain.

Fig. 2.—Posteroanterior view of skull showing erosion of the left mandibular ramus and the coronoid and condylar processes.

On June 20, 1956, the patient reported again for a checkup, stating that his general condition had improved but that he was rather worried about a swelling which had appeared in the region of his left parotid gland. He was advised to report back to the surgical outpatient department; before he could do so, however, the swelling increased over a period of forty-eight hours and became extremely painful, and there was difficulty in opening his jaws. He was therefore admitted to the hospital on June 23, 1956, with a diffuse inflammatory swelling of the left parotid region, suggestive of an acute suppurative parotitis.

The patient had pyrexia and treatment consisting of systemic penicillin every six hours and short-wave diathermy was commenced. As a result of this, his general condition improved and the facial swelling partially resolved, leaving a firm preauricular swelling, 1½ by 1 inch in size, which was tender on pressure. Examination at this stage showed good opening of the jaws, with marked deviation to the left of the midline. Paresthesia in the left mandibular nerve distribution was noted. Cervical lymph glands were palpable on the left side of the neck.

Radiologic examination of the skull and jaws showed a well-marked soft-tissue swelling in the region of the left parotid gland, with gross erosion of the underlying mandible. There was total loss of the proximal portion of the ascending ramus, and the coronoid and

condylar processes were also similarly affected. The mandibular bone adjacent to the lesion showed an irregular margin, suggesting malignant osteolytic change. These findings suggested the presence in the left mandible of either a primary invasive tumor or a destructive lesion metastasizing from the primary tumor of the colon.

A radiotherapist, seen in consultation, expressed the view that this was almost certainly a secondary tumor and that, as the original neoplasm had been of colonic origin, such a secondary tumor of bone was unlikely to prove radiosensitive. Drill biopsy was considered but was rejected in view of the inability to provide effective treatment by either deep x-ray or surgery. As the acute inflammatory reaction had subsided, the patient felt much more comfortable, and he was discharged from the hospital at the beginning of July, 1956.

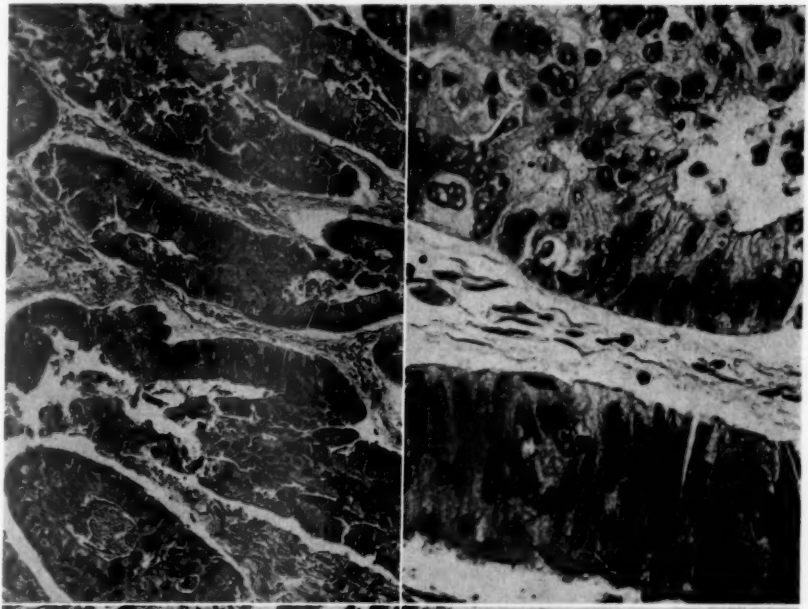
Fig. 3.—Lateral roentgenogram of mandible.

He remained reasonably well until September, 1956, when he was again suffering considerable pain in the jaw and had to be readmitted to the hospital. By this time, the size of the jaw swelling had increased considerably. Abdominal examination did not reveal evidence of ascites or liver enlargement. The patient required heavy sedation with morphine and Omnopon; his general condition slowly deteriorated, and he died on Nov. 2, 1956.

Autopsy.—The body was that of a wasted, elderly man. There was a swelling over the left parotid region which, on section, had the appearance of a necrotic carcinoma. The pleural cavity showed numerous old-standing adhesions and deposits of carcinoma in the lower lobe of the right lung. The myocardium was soft and showed evidence of old rheumatic disease of the aortic and mitral valves. In the abdominal cavity, there was no local recurrence at the site of operation, but the liver was somewhat enlarged although normal in color. On section, several deep-seated secondary carcinomatous deposits were revealed.

Histologic Examination.—Sections of the primary tumor of the colon and of the secondary deposit in the ascending ramus of the mandible were studied. The sections from the ascending colon showed a columnar-cell carcinoma of an intermediate grade of differentiation

A. Fig. 4. *B.*



A. Fig. 5. *B.*

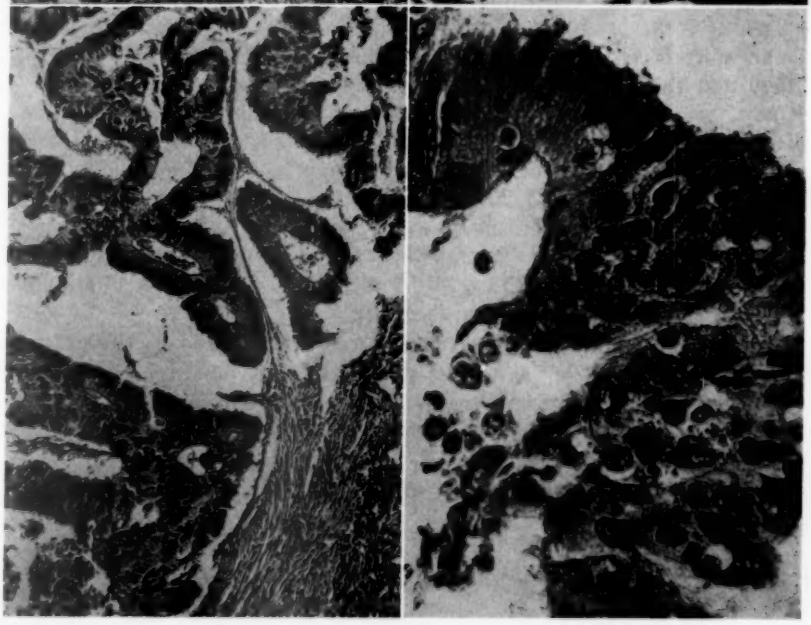


Fig. 4.—Primary growth. *A*, Low-power view; *B*, high-power view.
Fig. 5.—Metastasis. *A*, Low-power view; *B*, high-power view.

infiltrating almost completely through the wall of the cecum. The only gland apparently involved was one from beside the ligature on the most distal blood vessels of the colon. Postmortem sections of the secondary deposit in the mandible showed well-differentiated adenocarcinoma, which had eroded through the substance of the mandible and spread into surrounding soft tissues.

DISCUSSION

Cohen,[1] in an excellent recent survey of secondary tumors of the mandible, quotes Sharp and associates,[3] who state that the most common primary sites of mandibular metastasis are the breast, thyroid, and lung. Moorman and Shafer[4] found the most common primary sites to be the breast, thyroid, ovary, lung, kidney, and rectum.

Stern and Shephard,[5] in a survey of metastatic carcinoma of the mandible, stated that the lesions were generally primary, involving the tongue, lips, and cheek. They found few references in the literature to metastatic carcinoma of the jaws. Perusal of their classified table of recorded metastatic cases shows that from a total of forty-four cases, the primary tumor was in the thyroid in thirty-three cases, the breast in five cases, the prostate in three cases, the lung in two cases, and the rectum in one case.

Byars and Sarnat[6] reported two cases of secondary mandibular tumors. One of these tumors, which occurred in a 57-year-old woman, arose from a primary breast tumor, and the other, in a woman aged 55 years, arose from a primary tumor of the thyroid. Kronfeld and Weinmann[7] described an adenocarcinoma in the jawbone which showed extensive formation of new bone; this arose from an adenocarcinoma of the prostate in a man of 63 years. Salmon and Darlington,[8] in an article entitled "Rare Malignant Tumors of the Jaw," described eleven cases of metastatic tumors of the jaws, one of which is of interest because it arose from the gastrointestinal tract. The primary lesion was a carcinoma of the stomach in a 49-year-old man, and there were metastases to the liver, lungs, and mandible.

It will thus be seen that mandibular secondary tumors arising from primary sites within the gastrointestinal tract are relatively rare. This could be claimed particularly for the case described in this article, in which the ascending colon was the site of the primary tumor.

No other reference to this primary site can be traced as having metastasized to the mandible, although Thoma[9] cites a case reported in 1936 by Humphrey and Amos, in which metastasis was found in the gingival tissue from a primary adenocarcinoma of the colon occurring in a 33-year-old man. The lesion, which was pedunculated, soft, reddish, and ulcerated, occurred near the maxillary first molar and had not involved bone. There were metastases to the lungs and liver also.

SUMMARY

1. A case of primary adenocarcinoma of the ascending colon giving rise to osteolytic metastasis in the mandible has been described.

2. The literature relevant to metastatic tumors of the mandible has been reviewed, showing the gastrointestinal tract to be an uncommon primary site and a primary tumor in the ascending colon a rarity.

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HISTOCHEMICAL ANALYSES OF WHITE LESIONS OF THE MOUTH

I. The Basement Membrane

Lester R. Cahn, D.D.S., Leon Eisenbud, D.D.S.,** and Melvin N. Blake, D.D.S.,*** New York, N. Y.*

THIS is a report of findings with reference to form and continuity of the basement membrane in 100 white lesions of the oral mucosa. In this number were lesions exhibiting varying degrees of epithelial hyperplasia, keratosis, and parakeratosis. Occasional sections showed dyskeratosis and disorientation, and some demonstrated the histologic characteristics usually ascribed to lichen planus, discoid lupus, and white sponge nevus. Fifteen cases of undoubted carcinoma served as controls.

Since the specimens analyzed represent 100 successive accessions in the white lesion category, the relative incidence of the various entities, classified on the basis of sections stained with hematoxylin and eosin, may be of interest:

Epithelial hyperplasia	7
Epithelial hyperplasia with keratosis	35
Epithelial hyperplasia with parakeratosis	40
Epithelial hyperplasia with dyskeratosis	1
Lichen planus	9
Discoid lupus	2
White nevus	6

The basement membrane is defined electromicroscopically as an amorphous layer, possibly of a mucopolysaccharide character, to which fine collagenous fibers staining as reticulum adhere.

It may be assumed that the basement membrane is permeable to intercellular fluids which diffuse through this structure to provide an interchange of nutrients and waste products. The cytoplasmic processes of basal cells pass across the basement membrane to join reticular fibers in the stroma and provide anchorage for the epithelium.

From the Oral Pathology Laboratory of the Mount Sinai Hospital.

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*Attending Pathologist (Oral Pathology), Mount Sinai Hospital.

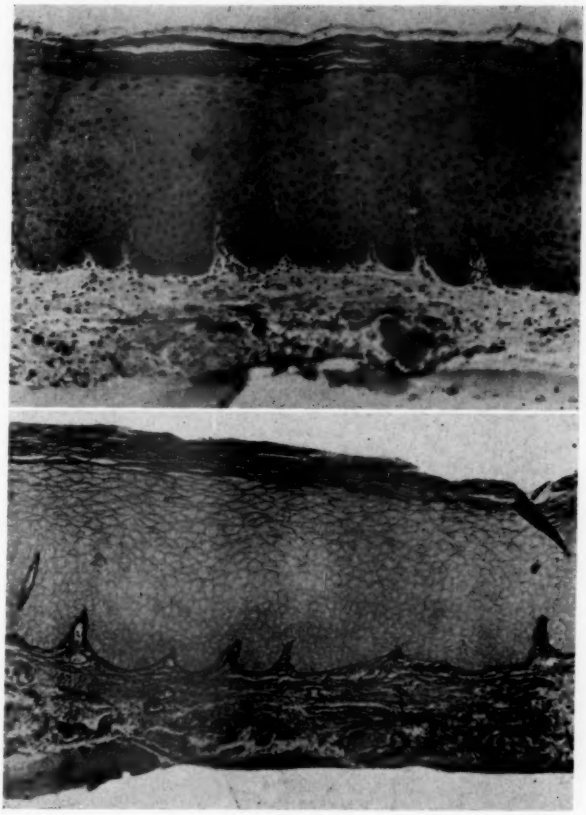
**Associate Attending Pathologist (Oral Pathology), Mount Sinai Hospital.

***Research Assistant (Oral Pathology), Department of Pathology, Mount Sinai Hospital.

Impermeability to large foreign particles is demonstrated by the fact that dyes injected intradermally beneath a subepidermal bulla will not appear in the fluid of the bulla, although it spreads rapidly through the dermis.[1]

With reference to the penetrability of the basement membrane by cells, histologic evidence seems to indicate that neutrophils (for example, in psoriasis) may cross the basement membrane and find their way into the region of the

A.

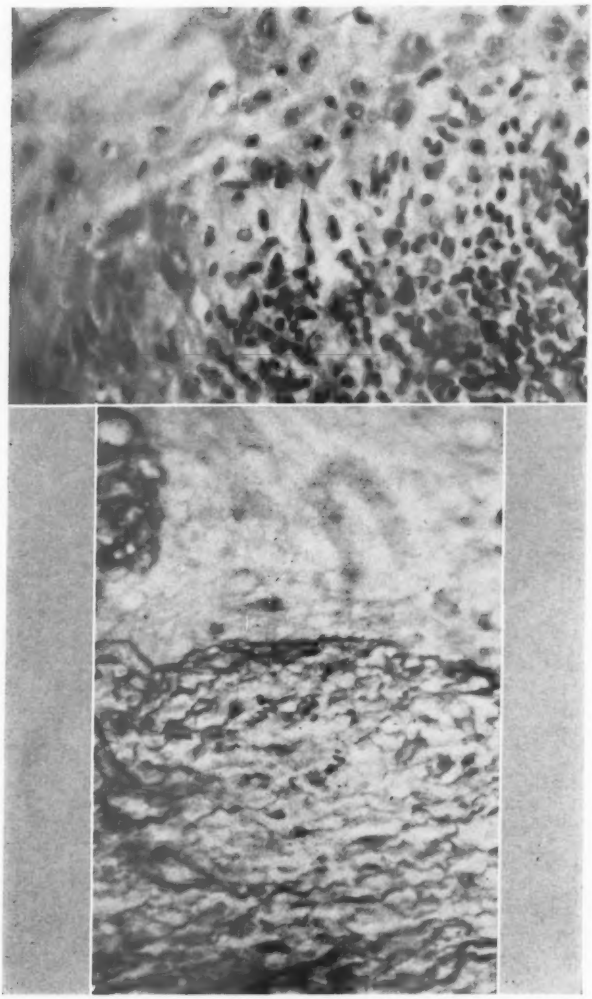


B.

Fig. 1.—Epithelial hyperplasia with parakerotosis. *A*, Hematoxylin and eosin stain shows a clear delineation between the epithelium and stroma, but the basement membrane cannot be seen. *B*, With P.A.S., the basement membrane stains strongly.

stratum germinativum and prickle-cell layers. In the pigmented nevus, cells, probably of basal origin, drop through the basement membrane and are found in nests in the underlying corium. Although note may be made of these special instances of cellular transgression, the basement membrane in general seems to be a limiting structure which marks the border of the epithelium even in the presence of extensive overlying hyperplasia.

A.



B.

Fig. 2.—Lichen planus. *A*, With hematoxylin and eosin stain, the basal layer is vague and washed out and is invaded by the subepithelial infiltrate. *B*, P.A.S. stain discloses a basement membrane that stands out vividly.

Thus, the continuity or lack of continuity of the basement membrane may be of significance in the containment of epithelial downward growth. Routine hematoxylin and eosin stains do not demonstrate this structure, however, even though a clear delineation between the epithelium and the stroma may

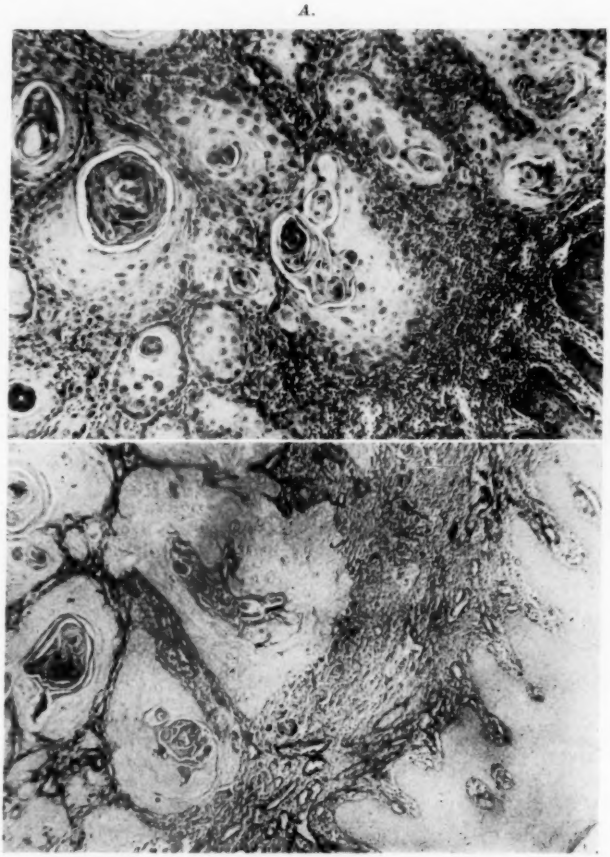


Fig. 3.—Squamous-cell carcinoma. *A*, Sections stained with hematoxylin and eosin show nests of malignant cells, keratin, and parakeratin pearls. *B*, With P.A.S. stain, the basement membrane is found to be missing.

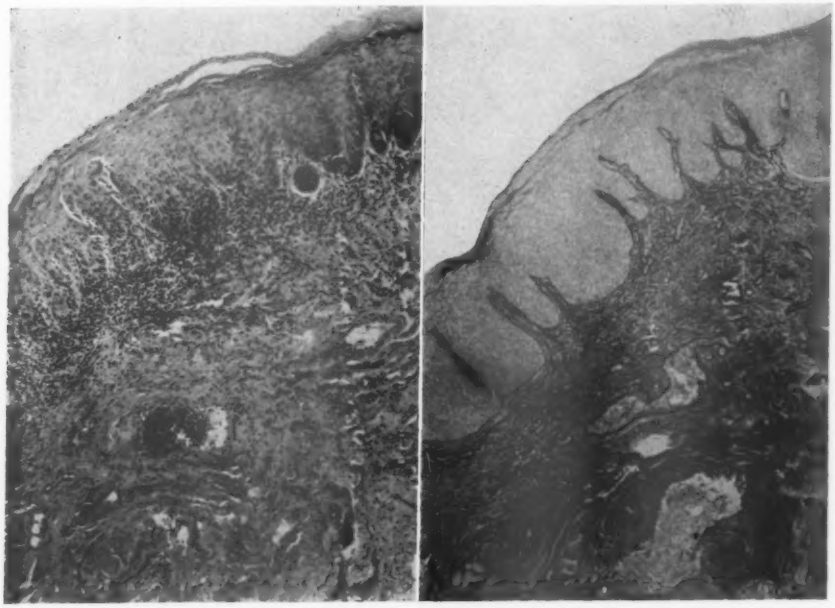
be indicated by a continuous and well-defined basal layer. Often an infiltration of inflammatory cells obscures this zone. In disoriented epithelium, the barrier to epithelial incursion cannot be assessed accurately.

With P.A.S. stain, the basement membrane can be delineated beautifully (Fig. 1). It assumes a vivid magenta color.

It is interesting to compare sections of lichen planus stained with hematoxylin and eosin and with P.A.S. stain. With hematoxylin and eosin, the basal

layer is vague and washed out and is invaded by the subepithelial inflammatory infiltrate (Fig. 2, *A*). It appears that there is no basement membrane. Sections from the same block stained with P.A.S., however, disclose a basement membrane that clearly stands out (Fig. 2, *B*).

In frank squamous-cell carcinoma, the basement membrane no longer takes on a heavy stain. In the fifteen cases studied, it was found to be missing in ten and vague in the remaining five (Fig. 3, *A* and *B*).

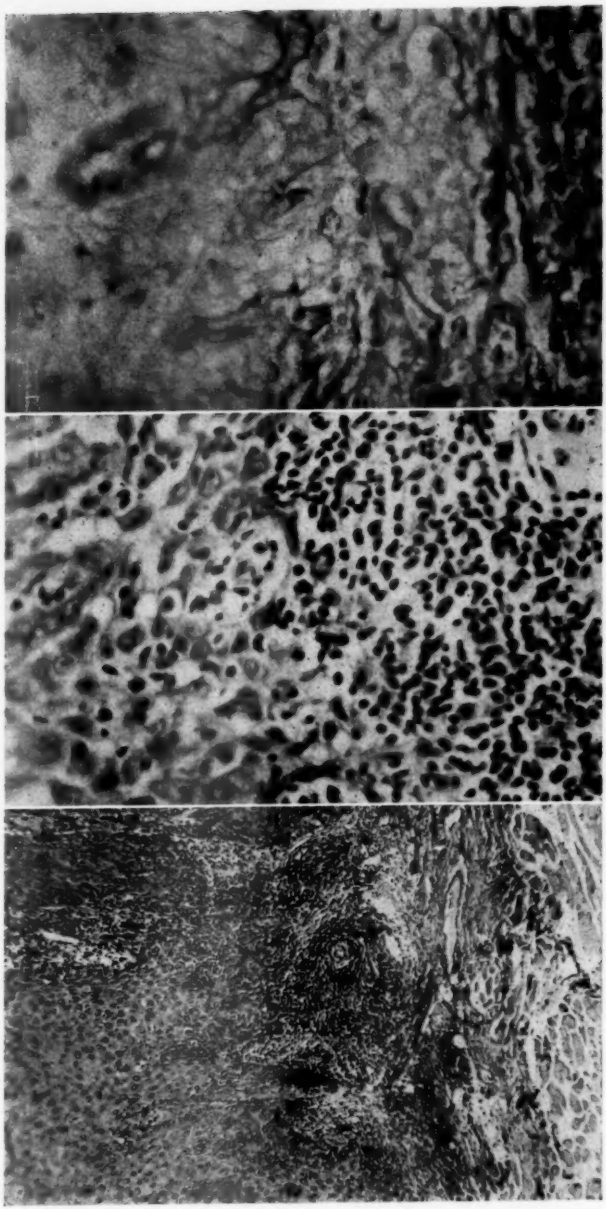


A. *B.*

Fig. 4.—Epithelial hyperplasia with disorientation. *A*, Hematoxylin and eosin stain shows well-defined basal layer. *B*, With P.A.S., the basement membrane stains brightly.

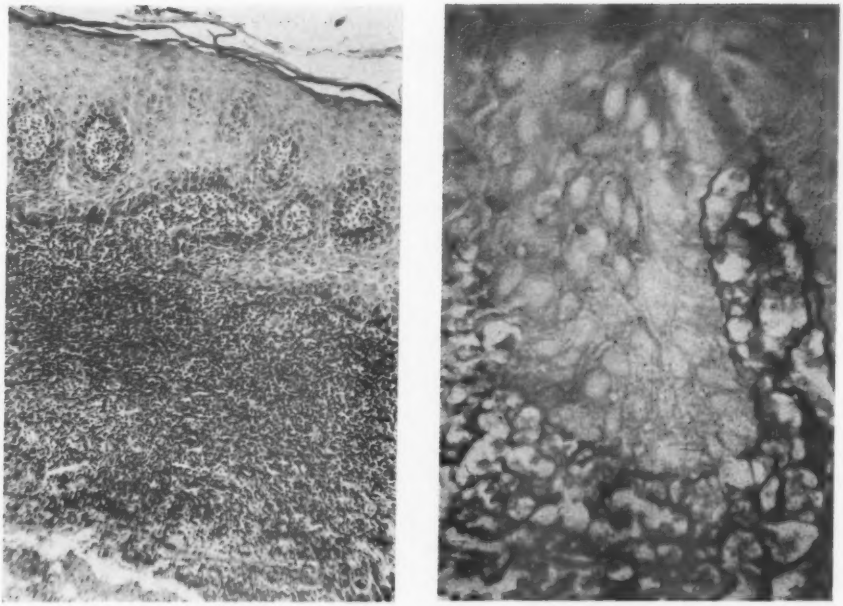
In this series of 100 white lesions it was possible to demonstrate the basement membrane in each case. In several instances of disorientation, the basement membrane stained brightly and was intact (Fig. 4, *A* and *B*). On the other hand, in some instances of disorientation and dyskeratosis, the basement membrane was vague and appeared to be undergoing dissolution (Fig. 5, *A* to *C*). In one case of epithelial hyperplasia with a lichenoid infiltration the membrane was vague and interrupted and in this case neoplastic changes later developed (Fig. 6, *A* to *C*).

These findings concerning the basement membrane lead to the tentative conclusion that there is a substance, possibly enzymatic in nature, elaborated by malignant epithelial cells, that dissolves the P.A.S.-positive material in the basement membrane. This same substance may be present in potentially malignant cells, such as are found in areas of disorientation without invasion



A. *B.* *C.*

FIG. 5.—Epithelial hyperplasia with disorientation. *A* and *B*, Hematoxylin and eosin stain shows a well-defined delineation between the epithelium and stroma. C, With P.A.S. stain, the basement membrane appears to be undergoing dissolution.



A. *B.*

Fig. 6.—*A*, Section of epithelial hyperplasia with lichenoid infiltration in which neoplastic changes later developed. (Hematoxylin and eosin stain.) *B*, P.A.S. stain shows the basement membrane to be vague and interrupted.

(leukoplakia, intraepithelial carcinoma, etc.). Furthermore it is tentatively concluded that the P.A.S. stain may make it possible to distinguish potentially malignant epithelium from benign epithelium and maybe to separate benign white lesions from potentially malignant ones.

The excellent sections used in this study were prepared by Mrs. Sarah Spector, Research Histology Technician, Mount Sinai Hospital.

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ENDODONTICS

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ROOT CANAL DRESSINGS

Their Usefulness in Endodontic Therapy Reconsidered

Samuel Seltzer, D.D.S., I. B. Bender, D.D.S.,** and Irving J. Kaufman, D.D.S.,*** Philadelphia, Pa.*

INTRODUCTION

DURING endodontic therapy various medications are used as dressings in the root canal, their functions presumably being to eliminate or reduce microbial flora, prevent or lessen pain, reduce inflammation, or stimulate repair. In this article we will critically consider whether some of the commonly used drugs do, in fact, accomplish these purposes. In addition, we will describe some of our own experiments dealing with the effects of several substances on inflammation and repair.

Elimination or Reduction of Microbial Flora.—Most of the drugs used in endodontic therapy are claimed to be potent antibacterial and/or antifungal agents. In comparative studies of standard germicides and antibiotic mixtures, claims are made for the superiority of one medicament over another on the basis of the number of treatments required before negative cultures can be obtained.[5, 14] This preoccupation with the effects of drugs on microorganisms has diverted attention from a more pertinent endodontic problem—the effects of such drugs on tissues. Most endodontic drugs do eliminate or reduce microbial flora, but they also have other effects. The commonly used germicides, injudiciously applied, are irritating to apical tissue.[7, 10] A drug which kills bacteria may also kill vital tissue; in many instances the drug does more damage than the microorganism ("the cure is worse than the disease").

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*Associate Professor of Oral Pathology and Oral Histology, School of Dentistry, University of Pennsylvania.

**Associate Professor of Oral Medicine, School of Dentistry, University of Pennsylvania.

***Dental Staff, Einstein Medical Center, Northern Division.

Some antibiotics are less irritating to tissues but have other undesirable effects. Penicillin and other antibiotics may provoke dangerous allergic reactions at the time of treatment, or the patient may build up a sensitivity to the drugs and thus be deprived of their benefits when he needs them more urgently.

Successful endodontic therapy does not necessarily require the use of drugs. Good results, with apical repair, have been reported by investigators[1, 15] who relied solely on mechanical or chemomechanical cleansing and débridement of the root canal, a process which physically removes large numbers of organisms together with inflamed necrotic tissue and inflammatory products. The débrided root canal presumably provides favorable conditions for the body's defenses and thus for repair of the apical lesion.

The body's defenses are often adequate in spite of incomplete sterilization. This has been demonstrated occasionally when root canals have been filled after failure to obtain negative cultures. In some instances the results were nonetheless successful. These cases cast some doubt on the importance of adhering rigidly to the commonly accepted rule that a negative culture must be obtained before a root canal is filled, but they should not be construed as proof that repair occurs regardless of the presence of infection. A study is currently under way[3] to determine scientifically whether or not repair of the apical tissues can occur even though the root canal is not sterile. The results will be reported later.

Prevention or Reduction of Pain.—The literature contains a few reports on the use of drugs within the root canal to reduce or eliminate pain during endodontic therapy. Sommer, Ostrander, and Crowley[14] reported that glycerite of iodine was effective in the control of pericementitis. The pain-relieving action of this substance, they thought, might be related to its hygroscopic properties, although they could offer no definite explanation.

Wolfsohn[15] claimed that hydrocortisone markedly lowered the incidence of pain in apical periodontitis; he thought that the drug acted by reducing inflammation in the tissues surrounding the disease process. His results may be questioned, for he did not use a double-blind technique with placebos, and he failed to treat his control and experimental groups alike. Also, the 71 per cent incidence of pain in his control group does not agree with clinical experience.

Ostby[9] reported great success in relieving pain and reducing edema during endodontic therapy with sulfathiazole. Seltzer, Bender, and Ehrenreich[11] made an extensive investigation of the use of sulfonamides and other compounds for the reduction of pain during endodontic treatment. They found little difference between sulfonamides and placebos as far as incidence and severity of pain were concerned and a slight, although not statistically significant, advantage for sulfonamides with respect to duration of pain. These investigations also disclosed that about 40 per cent of all patients experience some degree of pain or discomfort during the period of endodontic therapy.

Although one might have expected a correlation between pain and the presence of infection in the root canal, actually no differences in pain response could be found in patients with respect to positive or negative cultures.

Reduction of Apical Inflammation.—The purpose of endodontic techniques for eliminating microorganisms and/or their products is to reduce inflammation. Removal of necrotic tissue and tissue-breakdown products through débridement and dressing of the canal is also thought to help accomplish this end. There is some question whether the adjunctive use of drugs is effective in reducing inflammation. In experiments to be described in this article, the drugs tested actually increased the inflammatory responses of the apical tissues. Most drugs used to eliminate or reduce microorganisms in the canal also injure the tissue.[14] Irrigants have been used in the canal to dissolve necrotic tissue, pus, or tissue debris, but the irritating qualities of some of the solutions thus employed are well known.[10] Similarly, digestive enzymes have been used clinically in endodontics,[2, 4] but they, too, cause some undesirable side effects. Further investigations are necessary to determine whether irrigants reduce or actually increase irritation.

Stimulation of Repair Processes.—In endodontics, drugs may be used to stimulate fibroblastic, osteoblastic, or cementoblastic activity or to increase the mucopolysaccharide content in repair tissue.

Matsumiya, Suzuki, and Takuma[7] used calcium hydroxide within the root canal to treat apical inflammation. Photomicrographs showed not only that the apical inflammation subsided, but also that cementoid tissue formed (within forty-eight days in some instances), thereby closing off the apical foramen. In other teeth, where canals were filled with calcium hydroxide, active regeneration of the alveolar bone around the root apex was demonstrated within forty days. The investigators concluded that the calcium hydroxide closed the root canal not by simple mechanical means but by accelerating the natural healing processes of the apical tissues.

Bonelike closure has not, as yet, been demonstrated by any valid study involving examination of serial sections. Heterotopic ossification or calcification in the soft tissues of rats following subcutaneous implantation of calcium hydroxide pellets has been reported by Mitchell and Shankwalker,[8] but these investigators made no studies of apical tissue reaction to calcium hydroxide.

Kuttler's[6] clinical investigations led him to conclude that dentinal shavings, implanted in the terminal portion of the root canal, stimulate more rapid regeneration of apical tissues and formation of new cementum. His conclusions, however, were not supported by histologic evidence.

Drugs used to increase the mucopolysaccharide content in repair tissues have not yet been employed in endodontics.

In previous investigations,[12, 13] we observed an increased activity in the formation of dentine matrix following the application of alkaline phosphatase to pulp exposures. The formation of matrix was abundant around dentine chips which the burr had pushed into the pulp during the process of exposing the pulp. If alkaline phosphatase could stimulate pulp tissue to

form dentine matrix, perhaps it could similarly cause apical pulp tissue to form dentine or apical periodontal membrane to form cementum. Apical ramifications might thus become obliterated through "physiologic closure" of the apical foramina. One of the purposes of the following study was to determine whether or not apical repair could be stimulated by calcium hydroxide, alkaline phosphatase, albumin, or saline solution. Another purpose was to demonstrate the sequence of events which occur during the formation of a periapical lesion in the course of endodontic therapy.

PROCEDURE

A young, healthy dog, approximately 1 year old, was placed under general anesthesia. The posterior teeth of the lower left quadrant (lower left first, second, third, and fourth premolars) were then opened through the biting surfaces, and the pulps were exposed. The pulps of each of these teeth were extirpated with sterile broaches, and the canals were reamed and filed to approximately the size of a No. 6 reamer. Bleeding was controlled with sterile paper points. Dentine chips were purposely left within the confines of the canal, and presumably some were pushed into the connective tissue of the apical foramina. Irrigants were not used. Solutions or suspensions of various medicaments in sterile distilled water were introduced into the canals of each tooth as follows: lower left first premolar, calcium hydroxide, 30 mg.; lower left second premolar, alkaline phophatase, 5 mg.; lower left third premolar, albumin, 5 mg.; and lower left fourth premolar, sterile saline, 0.5 ml. In order to seal the canal from the oral cavity, a small pledget of sterile cotton was inserted into the pulp chamber over the orifices of the canals and the cotton, in turn, was covered with an amalgam filling.

The same procedures were carried out the following week on the teeth of the lower right side and in successive weeks on the teeth of the upper left side and the upper right side. During the fifth week, the dog was killed by perfusion with fixatives. Following decapitation of the animal, the teeth were removed from the jawbone by means of a band saw, decalcified, and embedded in paraffin. Slides of the tissues were stained with hematoxylin and eosin for serial microscopic examination.

RESULTS

One week after pulp extirpation, and after reaming, filing, and medication of the root canals, the responses observed in the apical tissues of each of the teeth were about the same, regardless of the kind of dressing placed within the canal. The early changes, then, seem to be the consequences of the pulp extirpation, reaming, and filing, and not related to the kind of medicament used. In the apical foramina, evidence of an acute inflammatory response could be observed. The connective tissue was heavily infiltrated with polymorphonuclear leukocytes. Dentine chips, dead pulp cells, and debris were found in the main canal. Within the apical periodontal membrane and the

marrow spaces of the bone there were dense collections of polymorphonuclear leukocytes and erythrocytes and a profuse edema; that is, an acute inflammatory response had occurred.

In one of the teeth that had been treated with alkaline phosphatase, there was an accidental perforation near the apical third of the tooth root. Dentine chips in abundance had been pushed into the remaining apical pulp tissue. A profuse quantity of new matrix was found around these chips, tending to weld them together. No evidence of new matrix activity was found in the remaining apical pulp tissues of the other teeth.

Within *two weeks* (that is, in teeth which had been treated two weeks before the dog was killed) the acute inflammatory response was even more pronounced; the collections of acute inflammatory cells were much denser and seemed to have infiltrated the marrow spaces in greater depth. The fibers of the apical periodontal membrane were now separated by the edema and the dense collection of inflammatory cells. Bone resorptions occurred simultaneously at sites distant from the apical foramina, and osteoclastic activity was evident there. Apparently, space was being created for the developing granuloma. Blood vessels within the periodontal membrane were dilated and engorged with neutrophils. The periodontal fibers were swollen and disaggregated.

By the end of *three weeks* differences in inflammatory response were becoming apparent. They were related to the kind of medication that had been put into the canal dressing. Thus, a tooth treated with albumin showed a fairly intense inflammatory response which was approximately the same as that found in a similarly treated tooth at the end of two weeks. Where alkaline phosphatase had been used, response was approximately the same as that produced by albumin, being limited to a small area immediately adjacent to the apical foramina. Collections of inflammatory cells were of about the same density as those found after two weeks. The amount of bone resorption was about the same as in the tooth treated with albumin. A small apical lesion was present.

The lesion found in the calcium hydroxide-treated tooth at the end of three weeks, however, was of much greater magnitude and intensity. The bone resorption had created a much larger space apically. The inflammatory cell infiltration was denser and necrosis of tissue had occurred, indicating suppuration. In other words, an acute apical dentoalveolar abscess had developed.

With saline solution, the apical lesion three weeks after treatment was, in general, about the same as that found at the end of two weeks; the amount of bone resorption was unchanged, cells indicative of inflammatory response were less densely distributed, and there was no evidence of new dentine or cementum matrix.

At end of *four weeks* the effects of the drugs had become more apparent. In the teeth treated with albumin four weeks previously, the inflammatory

response and the size of the apical lesion were about the same as in the tooth representing effects three weeks after treatment.

In the teeth with alkaline phosphatase-treated root canals, the apical inflammatory response was tending to dissipate. Collagenous material was found in some of the apical ramifications, but this did not necessarily indicate that formation of collagen fibers in the apical region was due to stimulation by alkaline phosphatase.

In canals treated with calcium hydroxide, an acute apical abscess was still present; that is, abscesses were found in two teeth representing, respectively, effects three weeks and four weeks after treatment. This would indicate that the calcium hydroxide was quite irritating to the apical tissues.

Canals treated with sterile saline solution showed the least apical inflammatory response at the end of four weeks. The lesions were confined to a small region and were tending to dissipate. No further bone involvement was evident. The occurrence of new cementum was not discernible in the apical foramina. As judged by a comparison with the saline-treated controls, it seems that the inflammatory response following pulp extirpation, reaming, and filing becomes more violent after application of some medicaments.

DISCUSSION

Debris, dentine chips, dead and live pulp cells, and other material were trapped along the walls of the root canal during the process of extirpating the dental pulp and reaming and filing the canal. The apical tissues reacted to this extirpation, reaming, and filing by becoming acutely inflamed. The presence of albumin, alkaline phosphatase, calcium hydroxide, or saline solution as a dressing in the root canal had no apparent effect on the response of apical tissues during the first two weeks. The inflammatory reaction began to subside by the third week. Differences in intensity of inflammatory responses were discernible three weeks after the application of medicaments. The least amount of inflammation was produced by sterile saline solution. The most intense inflammatory response followed the application of calcium hydroxide.

Similarly, Mitchell and Shankwalker[8] found that a calcium hydroxide–gelatin mixture, put into experimentally produced wounds of the tibia in Cynomologous monkeys, caused an inflammatory and foreign-body giant-cell reaction, and they observed after forty-four days that healing was still delayed. They concluded that untreated control bone wounds healed better than treated bone wounds.

The apical inflammatory response appeared to begin simultaneously in the marrow spaces of the bone, where osteoclastic activity was found. Acute inflammatory cells were discernible, both within the marrow spaces and between the fibers of the periodontal membrane and the apical pulp tissue. It seemed that any stimulation of new collagen formation, apically, would be inhibited initially by the acute inflammatory response. There was some evidence that matrix formation may have occurred in the apical pulp tissue following the application of alkaline phosphatase. However, formation of new cementum

as an apical sealer could not be discerned. Further experiments in which the inflammation is allowed to subside before drugs are tested as stimulants of apical matrix formation and calcification are necessary.

Although inflammation seems to be aggravated by the presence of certain drugs in sterile root canals, perhaps microorganisms produce an even greater inflammatory response. Should such be the case, the use of sterilizing drugs might, on balance, be advantageous. Here again, further investigations are needed.

SUMMARY AND CONCLUSIONS

The practice of dressing root canals with drugs in order to control microbial flora, pain, inflammation, and tissue repair was critically discussed. Further evidence bearing on the effects of drugs used in endodontics was obtained through histologic examination of a dog's teeth which had been treated with calcium hydroxide, alkaline phosphatase, albumin, or saline solution at various intervals before the experiment was terminated. Tentative conclusions are as follows:

1. Drugs used in endodontics to reduce or eliminate microbial flora may cause tissue damage or sensitize the patient.

2. Further investigations are needed in order to find intracanal medicaments which would be effective in reducing pain.

3. Irrigants and many tissue solvents employed in endodontics cause inflammatory reactions and other undesirable side effects.

4. The apical tissue reaction following pulp extirpation and reaming and filing of the canal is slightest after the use of sterile saline solution and greatest after the use of calcium hydroxide.

5. Physiologic closure of the apical foramina by newly formed cementum is not observed, even four weeks after the intracanal instillation of calcium hydroxide, alkaline phosphatase, albumin, or sterile saline solution.

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RESEARCH

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AN AMPLIFIER–GRAPHIC RECORDING METHOD FOR MEASURING ENAMEL PERMEABILITY

Harold E. Brewer, Ph.D., D.D.S., and Joseph C. Muhler, D.D.S., Ph.D., Indianapolis, Ind.

In a previous article[1] a method for measuring enamel permeability was described. This consisted of observing the conductance of an aqueous phase within an enamel shell immersed in an electrolyte solution as a function of time of immersion. In order to increase the accuracy and the utility of this method, an amplifier–graphic recorder has been substituted for the conductance bridge used in the previous study. The amplifier–recording apparatus has the advantages of (1) allowing direct objective measurement of conductance, and thus of permeability changes; (2) providing a continuous, permanent record of conductance changes occurring during any desired interval of time; and (3) good sensitivity to minute conductance changes. In this article we will describe the apparatus and demonstrate its utility for measuring the changes in enamel permeability brought about by agents known to affect enamel permeability.

APPARATUS

The apparatus is shown in Fig. 1. The conductance cell (*A*) is fabricated from two test tubes of different diameters from which the bottoms have been removed. The tooth is cemented to the smaller test tube with Plicene cement. The larger test tube, when cemented to the smaller one, serves as a chamber in which solutions are placed so as to be in contact with the enamel surface. Two platinum wire electrodes, approximately 30 mm. long, stabilized by a plastic-covered cork, are placed in the smaller test tube and held in place with another plasticized cork.

The conductance cell is mounted on an oscillating platform (*B*). The electric motor (*C*) causes the platform to rotate the conductance cell 360

From the Department of Biochemistry, Indiana University.
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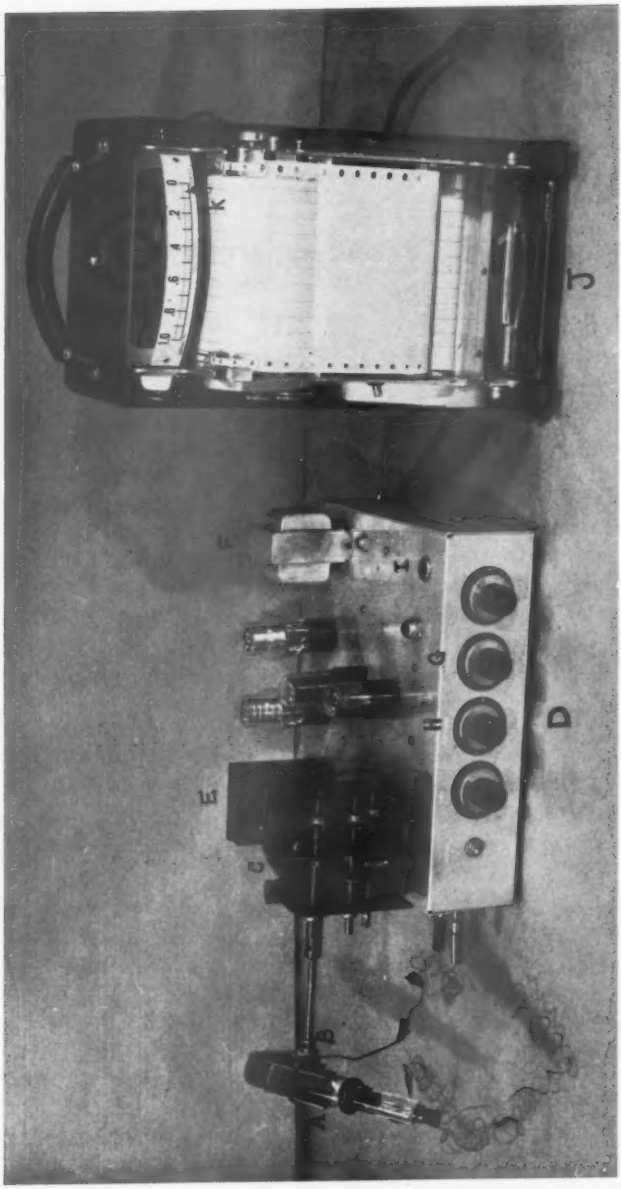

Fig. 1.—Photograph of the amplifier–graphic recorder apparatus used to measure enamel permeability. *A*, Conductance cell; *B*, oscillating platform; *C*, motor; *D*, amplifier; *E* and *F*, transformers; *G* and *H*, amplifier controls; *I*, calibration source; *J*, recorder; *K*, writing stylus.

degrees end-over-end once every ten seconds. A mercury switch is attached to the platform, so that it connects the cell electrically for five seconds of each ten-second period. The oscillating platform thus serves to (1) provide a homogenous solution around the electrodes, (2) minimize the generation of heat within the smaller test tube since current flows between the electrodes only during the time the mercury switch is closed, and (3) provide for the deflection type of recording in which the magnitude of each deflection indicates directly the conductance of the system at that time.

Leads from the cell electrodes are attached to the amplifier (*D*), and the unit is powered from a 115 volt outlet. Transformer *E* reduces line voltage for the motor (*C*), and transformer *F* is in the amplifier power supply. The range and sensitivity of the amplifier are adjusted by potentiometers (*G* and *H*). A calibration source is indicated at *I*.

The amplifier output (Esterline-Angus graphic ammeter Model AW, Esterline-Angus Co., Inc., Indianapolis, Indiana) activates the writing stylus (*K*) on the graphic recorder (*J*). The graph paper moves through the recorder at a constant speed. Each chart contains six deflections per minute of paper advance, each corresponding to a ten-second period of the oscillating platform.

The apparatus consistently detected and recorded conductance changes produced by as little as 3×10^{-6} molar sodium chloride solution. This was determined by measuring the relative conductance of twenty-four sodium chloride solutions ranging in concentration from 38 to 39,325 micromolar. The conductivity of the distilled water in which the electrodes were immersed was determined, and then the conductances of progressively more concentrated sodium chloride solutions were measured. The relative conductance units (RCU), which vary from a value of 1.00 when a 38 μm sodium chloride solution was used to a value of 34.50 when a 39,325 μm solution was used, indicate the amplitude of the writing stylus produced on the fifty-line recording paper.

It was shown previously[1] that the cement (Cenco Plicene No. 11441) used to attach the tooth to the conductance cell was impermeable to sodium and chloride ions during a seventy-minute period of immersion in a 2.5M sodium chloride solution. However, because the physical qualities of the different batches of cement, especially workability and flow, were found to differ and because the cement was to be exposed to sodium chloride solutions for longer time intervals, it was necessary to re-evaluate certain properties of this cement. A cement shell was prepared and attached to the conductance cell. The conductance of the distilled water within the shell was recorded while the shell was immersed first in distilled water and then in 2.5M sodium chloride solution for three hours. These records show that the cement was impermeable to sodium and chloride ions and, in addition, that neither the glass used to construct the conductance cell nor the cement itself was a source of ions which would interfere with permeability measurements. When the period of immersion was lengthened to twelve hours, similar results were obtained.

EXPERIMENT

Sections of enamel prepared from human teeth and from crowns of the mandibular cuspids of dogs were used in testing the amplifier-recording method of measuring enamel permeability. The human teeth were placed in distilled water immediately after extraction. One or several enamel sections were prepared from a single tooth by sectioning with a diamond disk during immersion in distilled water. The surface of the enamel was polished in a rubber cup with flour of pumice. The thickness of the twenty-five different sections varied less than 0.4 mm. The sections were cemented to the conductance cell, and the permeability of each section to 2.5M sodium chloride was measured after a control test in distilled water. The duration of immersion in each solution was three hours.

Mandibular cuspids were extracted in toto from mongrel dogs. The teeth were washed in distilled water and sectioned transversely about 2 mm. below the cementoenamel junction. The crowns were attached to the conductance cell as previously described after being polished with pumice by the method described for human teeth.

The permeability of contralateral mandibular cuspids from four dogs was determined by immersion in 2.0M sodium chloride solution for eight hours. The mandibular cuspids from three of these same animals and those from another dog were treated with a 1 per cent solution of sodium fluoride or a 2 per cent stannous fluoride solution and then reimmersed in 2.0M sodium chloride for varying intervals of time in order that the effect of fluorides on the permeability of the teeth might be studied.

RESULTS AND DISCUSSION

The data on the permeability of twenty-five sections of human teeth to 2.5M sodium chloride show a marked variability in enamel permeability of different teeth (Table I). These findings confirm our previous data[1] suggesting considerable random variability in enamel permeability. Sections of enamel which contained small carious defects are more permeable than other sections prepared from the same tooth. Similar findings have been reported previously by Beust,[3] who found that carious teeth were more permeable than noncarious teeth to alcoholic basic fuchsin. Beust attributed the increased permeability of the carious lesion to "a loose molecular structure." Although the age of the patient from whom the teeth were obtained was not known in all instances, enamel permeability appears to decrease with age, as previously reported by Atkinson[4] and Fish.[5] Measurement of the thickness of the enamel sections showed that this factor did not explain the variations observed in the permeability of different sections. Actually, some of the thinnest sections were completely impermeable to sodium and chloride ions.

The results obtained from measuring the permeability of contralateral mandibular cuspids from the same dog show that the total conductance change is directly related to the time during which the tooth is immersed

in the sodium chloride solution and indicate that there are wide variations in the permeabilities of different teeth. For example, the total conductance change in the right mandibular cuspid of dog A was only 1.8 RCU, whereas the right mandibular cuspid of dog C showed a conductance change of 42.8 RCU for the same time period. However, the permeabilities of contralateral cuspids from the same dog were similar. The similarity in the permeability of contralateral teeth has also been reported by Bergrren,[6] who compared the permeability of contralateral dog cuspids to dyes.

TABLE I. PERMEABILITY OF HUMAN ENAMEL SECTIONS TO SODIUM AND CHLORIDE IONS AS MEASURED BY THE AMPLIFIER–RECORDER METHOD

TOOTH SECTION	PATIENT'S AGE	RELATIVE CONDUCTANCE CHANGES*		THICKNESS OF SECTION (MM.)
		WATER	2.5M NaCl	
A	Unknown	0	0.0	0.92
B	Unknown	0	3.5	1.03
C	Unknown	0	42.0	1.27
D	Unknown	1.0	38.0	1.12
E	Unknown	0	2.0	1.11
F	Unknown	0	43.0	0.96
G	Unknown	0.5	22.5	1.03
H	Unknown	0	45.0	1.06
I	Unknown	0.5	44.0	1.37
J1	39	0.5	4.0	1.24
J2	39	1.5	1.5	1.03
J3†	39	0	40.0	—
K1	35	1.5	2.0	1.22
K2	35	0.5	0.5	1.28
L1	35	0.5	10.0	1.09
L2	35	1.0	8.0	1.09
M1	29	0	0.0	0.86
M2	29	0.2	1.0	1.04
N	36	0.5	3.0	0.90
O	50	0	0.0	1.03
P†	21	2.0	23.5	—
Q	27	0.3	12.0	1.16
R	Unknown	0.1	1.5	1.06
S	Unknown	2.0	12.0	0.98
T	Unknown	0.0	0.3	1.12

*During three hours.
†Carious section.

That the actual passage of ions through the tooth is measured by the amplifier–recorder was demonstrated by immersing one of the dog cuspids in three successive changes of distilled water following immersion in sodium chloride. These data are plotted in Fig. 2. It is interesting to note that soluble ions are present in the extracted tooth and that the movement of these into the pulp chamber is of detectable magnitude, as indicated by trial A. Also of interest is a comparison of the data from trials B and C. Trial B shows the effect on conductance of immersing the tooth in sodium chloride, and in this particular tooth increases in time and conductance are almost linearly related. The conductance changes in trial C are due to ions remaining in the tooth after immersion in sodium chloride, since the conductance changes that occurred are greater in trial C than in trial A. The significance of the apparently greater rate of conductance change in trial C, when the

TABLE II. EFFECT OF SODIUM FLUORIDE AND STANNOUS

TOOTH	FLUORIDE TREATMENT*	IMMERSION FLUID	RELATIVE CONDUCTANCE AT STATED					
			0	½	1	1½	2	2½
A	None	NaCl, 2M	1.0	19.0	40.0	50.0		
	SnF_2, 2%	NaCl	2.0	22.0	28.0	29.0	29.0	29.0
B	None	NaCl, 2M	0.5	2.2	5.5	12.2	15.6	21.0
	SnF_2, 2%	NaCl	1.0	2.2	3.3	4.3	4.8	7.2
C	None	NaCl, 2M	1.2	4.5	6.7	9.0	12.0	15.0
	SnF_2, 2%	NaCl	1.0	2.0	2.3	3.2	3.8	4.5
D	None	NaCl, 2M	0.6	1.0	1.3	2.0	2.8	3.8
	NaF, 1%	NaCl	0.5	0.6	1.1	1.2	1.2	1.3
E	None	NaCl, 2M	0.3	0.5	0.8	1.0	1.1	1.3
	NaF, 1%	NaCl	0.5	0.6	0.9	1.0	1.0	1.5
F	None	NaCl, 2M	1.3	2.0	3.0	3.8	4.6	5.5
	NaF, 1%	NaCl	0.5	1.1	1.2	1.4	1.5	1.8

*Duration of treatment = 10 minutes.

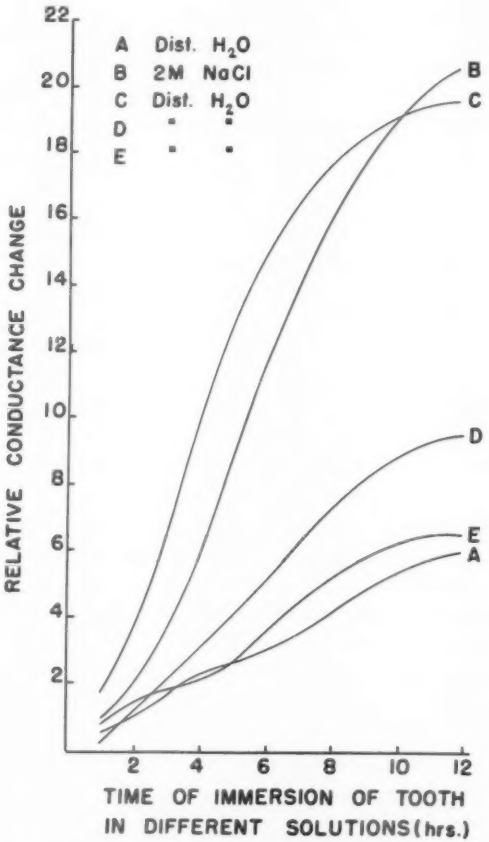

Fig. 2.—Graph illustrating the conductance changes within the pulp chamber of a dog's cuspid when immersed successively in distilled water (*A*); in 2M NaCl solution (*B*); and in three changes of distilled water (*C*, *D*, and *E*).

FLUORIDE ON PERMEABILITY OF MANDIBULAR CUSPIDS OF DOGS

HALF-HOUR INTERVALS DURING IMMERSION IN ELECTROLYTE SOLUTION										
3	3½	4	4½	5	5½	6	6½	7	7½	8
29.0	29.0	29.0	29.5	29.0	29.0	29.0	29.0	29.0	29.0	29.0
24.0	27.0	29.5	33.0	35.2	39.0	40.2	43.0	44.5	46.8	49.0
10.4	12.3	15.8	20.0	21.8	23.2	25.5	27.0	27.0	27.0	27.0
18.0	20.5	22.8	25.2	28.5	31.5	34.0	36.0	39.0	40.8	42.5
5.0	5.8	6.0	6.9	8.0	8.5	9.5	10.7	12.0	12.8	15.0
5.0	6.8	10.0	12.8	16.8	21.0	25.0	29.3	33.2	36.0	40.0
2.0	2.2	3.4	4.2	7.6	11.2	15.0	18.6	22.8	26.5	30.5
2.0	3.5	6.0	9.5	15.0	20.5	26.0	31.0	36.5	41.5	45.5
2.5	4.0	7.2	12.0	19.0	25.0	28.5	32.5	38.0	41.8	47.0
7.0	7.8	8.8	9.4	10.8	11.7	12.8	14.0	15.4	17.2	18.5
2.0	2.1	2.3	2.7	3.0	3.2	3.5	4.0	4.4	5.0	6.8

tooth was immersed in distilled water, than in trial B, when the tooth was immersed in sodium chloride, is not clear. The data from trial D indicate continued movement of ions into the pulp chamber of the tooth, whereas in trial E the total mass of ions which entered the tooth during its immersion in sodium chloride has been removed.

In order to demonstrate the utility of the method and to present data obtained through the use of these techniques which can be compared with previous studies in which different techniques were employed, a comparison of the antisolubility effectiveness of stannous fluoride was made. In a previous article[1] it was reported that the topical treatment of enamel with stannous fluoride resulted in a greater decrease in enamel permeability than similar enamel treatment with sodium fluoride. In the earlier investigation the enamel was exposed to the fluoride solution for four minutes and then immersed in sodium chloride for seventy minutes. In the present study the topical fluoride treatments lasted for ten minutes for each tooth, after which the teeth were immediately immersed in sodium chloride and the conductance was recorded for twelve hours. The data in Table II confirm the findings of the previous study and, more important, indicate (1) that the effectiveness of the fluoride solution in decreasing enamel permeability is a function of the time of exposure of the tooth to the fluoride solution and (2) that the permeability-reducing effect of stannous fluoride lasts for several hours.

SUMMARY AND CONCLUSIONS

An amplifier–graphic recorder for measuring enamel permeability has been described. Some characteristics of this apparatus have been demonstrated. Data obtained on the permeability of human and dog enamel have been presented to indicate the method's utility.

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BACTERIOLOGY

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FURTHER OBSERVATIONS ON THE IN VITRO ACTIVITY OF THE ANTI-HEMOLYTIC STREPTOCOCCUS FACTOR PRESENT IN SALIVA

Henry A. Bartels, B.S., D.D.S., Harry Blechman, D.D.S., M.A., and Gerald Wallach, B.S., New York, N. Y.

THE relative infrequency of infection by the beta hemolytic streptococcus subsequent to oral surgery procedures, extractions, root canal therapy, and periodontal treatments has been reported.[1] This finding led to an investigation to determine whether saliva contains a factor or factors which would inhibit the in vitro growth of beta hemolytic streptococci. It was found that freshly stimulated saliva exhibits an inhibitory action toward strains of beta hemolytic streptococci, Group A, under in vitro experimental conditions.[2] The factor is distinct from lysozyme, it is heat labile, and it is retained by the Seitz sterilizing filter. It appears to be associated with the oral microbial flora, and it is effective in inhibiting growth of both stock and recent beta hemolytic streptococcal isolates. Since the factor inhibits the growth of hemolytic streptococci in the presence of laked blood, potato juice, or lyophilized catalase and is active under anaerobic conditions, it was concluded that it is distinct from biogenic in vitro production of hydrogen peroxide. Recent studies[3] indicated that a similar factor associated with the oral microbiota inhibited the in vitro growth of *Clostridium tetani.*

In the present article we will report on additional findings associated with this antibacterial factor.

METHODS AND RESULTS

Experiment 1. The In Vitro Relationship Between the Numbers of the Oral Bacterial Population and the Test Organisms.—Freshly stimulated saliva was collected from several persons at various times and diluted serially from 10^{-1} to 10^{-9}. Duplicate pour plates were made with 1 ml. of each dilution.

From the Department of Microbiology and The Murry and Leonie Guggenheim Foundation, Institute for Dental Research, College of Dentistry, New York University.

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Upon solidification, the surface was streaked with the test organisms. The plates were incubated aerobically at 37° C. and observed at twenty-four and forty-eight hours for growth of the oral microbiota and the test organisms.

Results: Micrococcus lysodeikticus was inhibited in dilutions to 10^{-2}. The presence of numerous colonies of oral microorganisms did not restrain its growth. Counts of less than 3,000 to 4,000 colonies per dilution plate did not repress excellent growth of the beta streptococci on the surface of the medium. The presence of 4,000 to 8,000 colonies produced restricted growth, and colony counts above 8,000 caused complete inhibition of growth of beta streptococci.

Experiment 2. The Effect of Bacterial Products on the Activity of the Anti-Beta Hemolytic Streptococcus Factor.—Freshly stimulated saliva was added to aliquots of brain-heart infusion agar to give a 25 per cent concentration by volume, and plates were poured. A piece of sterile dialyzer tubing with a wall thickness of 0.00072 inch (Fisher Scientific Company), cut to the approximate diameter of the Petri dish, was placed on the solidified agar surface. The cellophane was then covered with a thin layer of sterile basal agar medium (overlay agar). Plates were prepared in triplicate and incubated for three days at 37° C., 25° C., and 5° C., respectively.

The agar layer covering the cellophane was then removed aseptically to sterile Petri dishes and streaked with suspensions of *M. lysodeikticus* and two strains of beta hemolytic streptococcus. The plates were incubated at 37° C. and observed after twenty-four and forty-eight hours for growth.

Results: The saliva-basal medium plates maintained at 5° C. showed no growth. The overlay agar from these plates permitted good growth of the test organisms. The plates maintained at 25° C. had numerous colonies of oral microorganisms. No colonies, however, appeared in the superimposed layer of agar. When this layer was removed to sterile dishes and streaked with the test organisms, inhibition of growth occurred.

The plates maintained at 37° C. had myriads of colonies of the oral microbiota in the saliva-agar layer. It was noted that microbial growth failed to pass the cellophane barrier into the overlay agar. The latter layer did not support growth of the beta hemolytic streptococcus strains.

Experiment 3. Determination of the Comparative Depth of Diffusion Through Agar of Lysozyme and the Anti-Beta Hemolytic Streptococcus Factor.—Freshly stimulated saliva was added to brain-heart infusion agar to give a 25 per cent by volume concentration. Plates were poured and allowed to solidify. Sterile dialyzing cellophane membrane (Fisher Scientific Company) was placed on the agar surface and overlaid with varying amounts of sterile brain-heart infusion agar.

Results: The manifest activity of salivary lysozyme decreases as the depth of the overlay agar increases. Its activity is not detectable under the given test conditions when the depth of the overlay agar is equivalent to 3 mm. In the case of the anti-beta hemolytic streptococcus factor, inhibition may be still noted with depths of overlay agar equivalent to 6 mm.

Experiment 4. Determination That the "Anti-Hemolytic Streptococcus Factor" is Distinct From Acid Production by Oral Microorganisms.—A bromcresol purple blood agar base (dextrose-free) was prepared by adding sufficient agar and indicator to Difco blood base medium to give final concentrations of 2 per cent and 0.02 per cent, respectively. Several 100 ml. aliquots were buffered with either 0.5 gram basic potassium phosphate and 0.2 gram monobasic potassium phosphate or with 1 gram calcium carbonate and adjusted to pH 7.2 to pH 7.4. Sufficient sterile 20 per cent dextrose was added to a number of each of the buffered basal media to give a final concentration of 1 per cent. A typical experimental run consisted of the basal medium with and without dextrose and the phosphate- and carbonate-buffered media with and without dextrose. Duplicate plates were prepared, one containing fresh saliva and the other containing heated saliva in 25 per cent concentration. Parallel surface streaks of *M. lysodeikticus,* beta hemolytic streptococcus, several Group A types, and *M. pyogenes* var. *aureus* were made on each of the plates. These plates were incubated for twenty-four to forty-eight hours at 37° C. and examined for growth and acid production.

Results: Acid production was manifest on the unbuffered 1 per cent fresh saliva-dextrose plates within twelve hours. The growth of the beta hemolytic streptococcus strains and *M. lysodeikticus* was inhibited, whereas *M. pyogenes* var. *aureus* grew unrestricted. On similar buffered (pH 7.2 to pH 7.4) dextrose plates, the same results were obtained. On control plates containing heated fresh saliva or without saliva, excellent growth of all test organisms was obtained.

Experiment 5. The Effect of pH on the Beta Hemolytic Streptococcus Factor and on Lysozyme Activity.—Brain-heart infusion agar (2 per cent) was prepared, dispensed in 100 ml. volumes in flasks, and adjusted to pH's of 6.0, 7.0, 8.0, and 9.0, respectively. Twenty-five per cent of saliva by volume was added to aliquots of the above. Pour plates were streaked with the test organisms.

Results: In the absence of saliva, good growth of *M. lysodeikticus* occurred through the pH ranges of 6.0, 7.0, 8.0, and 9.0. In the presence of saliva, *M. lysodeikticus* was inhibited completely at pH 6.0 and 7.0, grew poorly at pH 8.0, but grew moderately well at pH 9.0, indicating that salivary lysozyme is more potent in an acid or neutral medium.

The two beta hemolytic strains grew well at pH's 6.0, 7.0, and 8.0, but no growth occurred at pH 9.0. In the presence of saliva the beta hemolytic strains did not grow on the lots of medium at any of the adjusted pH's. The oral microbiota grew well in the pH range of 6.0 through 8.0 and moderately well at pH 9.0.

DISCUSSION

It is apparent that a relationship exists between the number of colonies of oral microorganisms growing in the depths of the saliva basal medium and the degree of growth suppression of the beta hemolytic streptococci on the

surface of this medium. When counts of less than 3,000 to 4,000 oral microbial colonies per dilution plate were obtained, unrestricted growth of the beta streptococci was observed. Counts of from 4,000 to 8,000 clones resulted in partial inhibition of surface growth, whereas counts of over 8,000 colonies caused complete inhibition of the beta hemolytic streptococcal surface growth. It is doubtful whether *Lebensraum,* or the physical presence of the colonies, is the answer, since the growth of the oral microbiota was essentially confined to the depths of the agar whereas the beta streptococci were inoculated upon the surface of the medium. It appears that the anti-beta hemolytic streptococcus factor is associated with the numbers of actively metabolizing microbial colonies.

The dialyzer cellophane membrane restricted passage of oral microorganisms into the overlay agar and permitted diffusion of the antibacterial factor. This activity was readily manifested when the overlay agar was removed and its surface streaked with strains of beta streptococci. Similarly, lysozyme passes the cellophane barrier, but its diffusibility was limited to approximately one-half the depth of that of the anti-streptococcus factor. Since anti-beta streptococcus activity on overlay agar from refrigerated saliva basal medium or from sterile parotid saliva plates was never observed, it was concluded that actively metabolizing microbial cells were necessary for the elaboration of this factor.

Certain diffusible microbial products have been shown to have antibacterial activity. Among these are such entities as hydrogen peroxide and acid production. Our previous results[2] indicated that biogenic hydrogen peroxide was not the active principle, since streptococcal inhibition occurred in the presence of catalase. Furthermore, data presented in this article indicate that the factor was distinct from the production of acids by microorganisms.

SUMMARY

The anti-beta hemolytic streptococcus factor was found to be intimately associated with the in vitro metabolism of actively growing oral microorganisms. A relationship was noted between the numbers of oral microbial colonies and the relative concentration of the inhibiting agent. The factor was found to be distinct from biogenic hydrogen peroxide and acid production.

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UNUSUAL DEPOSITION OF CALCULUS

Report of a Case

Jack Traiger, Captain, DC, USA

SUPRAGINGIVAL deposits of dental calculus occur most frequently, and in the greatest quantity, on the buccal surfaces of the maxillary molars and on the lingual surfaces of the mandibular anterior teeth. This has been explained on the basis of the proximity of these tooth surfaces to the orifices of the major salivary glands. The upper molars are adjacent to Stensen's duct, whereas the incisors approximate the openings of both the submaxillary and the sublingual glands.[1]

CASE REPORT

In March, 1959, a 14-year-old Negro girl came to the hospital dental clinic for an examination. She had no pain or discomfort at the time of the examination and was not aware of the presence of anything abnormal in her mouth. She was brought into the clinic by her mother, who stated that unless her mouth was cleaned up the patient would not be allowed to return to school.

Past Dental History.—Previous to this visit, the patient had not received dental care for several years and apparently had been quite negligent in the care of her mouth.

Medical History.—Aside from the common childhood infectious diseases, the patient's health had been good. Results of all laboratory tests were negative, and the family medical history was unrelated.

Oral Findings.—The gingival tissues, particularly in the mandibular anterior region, were acutely inflamed, edematous, and markedly hyperplastic. The patient's oral hygiene was extremely poor, and deposits (both supragingival and subgingival) were excessive. Other oral tissues, including the lips, the buccal mucosa, the floor of the mouth, and the palate, were normal in every respect. Radiographic examination revealed several carious lesions but no evidence of periapical infection (Figs. 1 and 2).

From the Oral Surgery Section, United States Army Hospital (Dental Clinic), Fort Jay, Governors Island, New York, New York.

DISCUSSION

The unusual feature of this case was the bizarre distribution of the calcareous deposits. The anterior teeth, both maxillary and mandibular, were heavily coated. The calculus even appeared on a great portion of the labial surfaces, an area which is usually relatively free of deposits. The deposits extended into the region of the mucobuccal fold, where they not only encased the clinical crowns of the teeth but also covered the attached gingiva and alveolar mucosa in the area.

Fig. 1.

Fig. 2.

Fig. 1.—Note the unusual heavy deposition of calculus in both the maxillary and the mandibular anterior areas.
Fig. 2.—Note the engorged, edematous, and hyperplastic gingival tissues.

The deposits were light brown in color and presented a pebblelike texture on the facial and proximal aspects.

SUMMARY

An unusual case in which extreme amounts of calculus were present in areas that normally tend to be free of such deposits has been described. The patient failed to return after her second visit to the clinic, and further examinations and treatment could not be carried out.

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SUBLINGUAL LIPOMA WITH ECTOPIC BONE FORMATION

Report of a Case

A. Fogle Godby, D.M.D., New Orleans, La., Peter B. Drez., D.D.S., Staten Island, N. Y., and James L. Field, D.D.S., Seattle, Wash.

THE lipoma is a tumor of adult fat cells which are grouped into lobules by vascular connective-tissue septa. It is commonly found in the subcutaneous tissues of the trunk, upper extremities, and neck but is encountered less frequently in the oral cavity. This fatty tumor grows slowly, but it may reach a large size. Malignant transformation seldom, if ever, occurs.[1] Bernier[2] observed that swelling is the most prominent symptom caused by this lesion and that only occasionally is there pain.

Simpson[3] reported finding seven oral lipomas as compared with fifty cases of carcinoma of the oral cavity in the same interval of time. Geschickter,[4] in reviewing 490 lipomas, found that only three occurred in the mouth. MacGregor[5] stated that of the various areas in the mouth, the sublingual region appears to be one of the most rarely involved. Thoma,[6] Pearl,[7] MacGregor,[5] Simpson,[3] Thoma and Robinson,[8] and Blake[9] have reported cases in which lipomas were found in the floor of the mouth.

The case reported here is considered unique for two reasons: (1) because ectopic bone was present within the tumor, and (2) because the base and the nutrient vessels lay in apposition to the periosteum at the genial tubercles.

In the differential diagnosis of a lesion of this type, many pathologic entities, such as dermoid cyst, salivary retention cyst, ossifying fibroma, myxoma, myositis ossificans, osteochondroma, and lipoma, must be considered.

CASE REPORT

The patient was a 54-year-old white man who had noted a swelling beneath the tongue for a period of about one year. This swelling had gradually increased in size and had finally enlarged to the point where it interfered with mastication and speech. It was not painful, and it was soft. The patient came to the United States Public Health Service Hospital at Staten Island, New York, for removal of the tumor.

The history was noncontributory, with one exception. The patient had once developed a pruritus after one injection of penicillin and was considered sensitive to that drug.

From the United States Public Health Service Hospitals in New Orleans, Louisiana, Staten Island, New York, and Seattle, Washington.

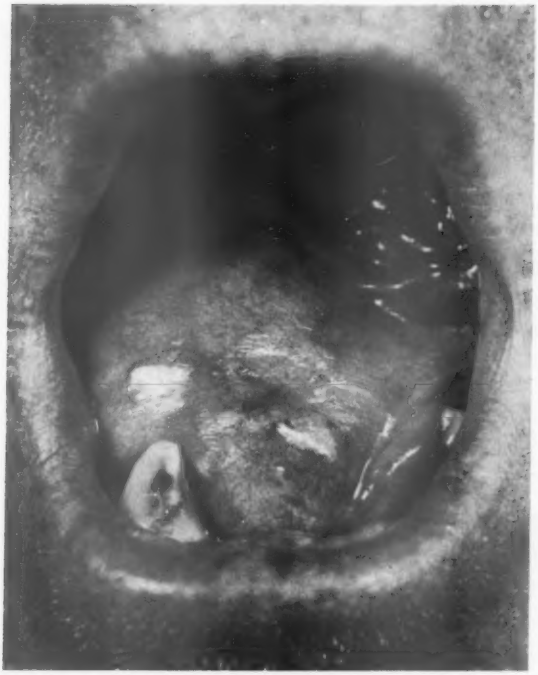

Fig. 1.—Preoperative photograph of the lipoma elevating the floor of the mouth above the occlusal surfaces of the teeth. The tongue is displaced laterally and posteriorly and is not visible in the photograph. The darkened structures in the upper right part of the mouth are the sublingual vessels.

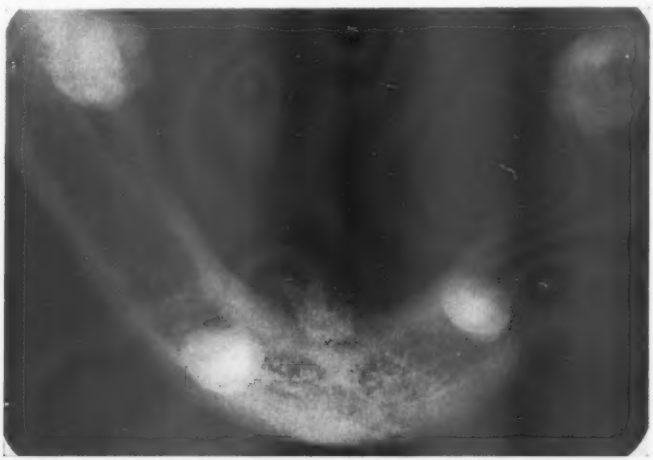

Fig. 2.—Occlusal view of anterior mandible and floor of the mouth. In the midline anteriorly may be seen an osseous projection. The radiologist's impression was that this lesion represented an osteochondroma.

Examination.—The patient, a well-nourished and moderately obese man, weighed 194 pounds. Examination of the mouth showed a large, soft, fluctuant mass beneath the tongue. The larger part of the mass was lying to the right of the midline. No submaxillary or cervical nodes were noted. Examination of the heart was negative except for a Grade 2-3 systolic murmur at the apex. There was a 2 plus enlargement of the prostate gland.

Laboratory findings, which included hematology, serology, and urinalysis, were essentially negative.

An electrocardiogram indicated that the systolic murmur noted on physical examination was probably functional in origin.

Chest x-ray findings were within normal limits. The radiologist's impression of the x-rays of the floor of the mouth was that the lesion represented an osteochondroma of the mandible.

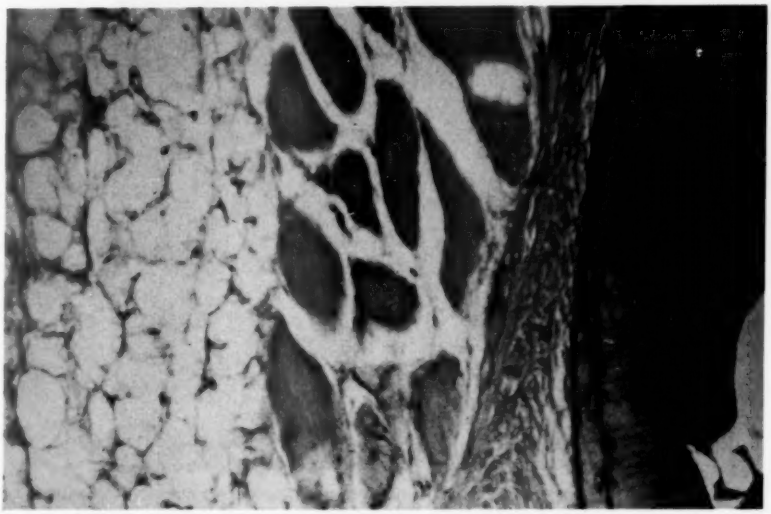

Fig. 3.—Photomicrograph of lipoma showing, from left to right, adult adipose tissue, striated muscle, fibrous connective tissue, and well-formed cancellous bone. (Magnification, ×40; reduced ⅓.)

Hospital Course.—The patient was admitted to the hospital on May 10, 1959, for excision of the sublingual mass. He was placed on prophylactic tetracycline therapy because of the possibility that the cardiac murmur might be organic in origin. On May 26, 1959, under nasal endotracheal anesthesia, the lesion was excised.

Operation: The operative site was prepared and draped in the usual manner. The two remaining mandibular cuspid teeth lying lateral to the tumor mass were extracted. A 5 cm. incision was made in the midline through the mucosa of the floor of the mouth from the crest of the mandibular ridge to the base of the tongue. The mucosa was reflected widely, and the thinly encapsulated, yellowish tumor herniated through the incision. The tumor contained many lobules which were seen to penetrate between muscle layers and other fascial spaces. The mass was bisected to facilitate removal and to avoid injury to adjacent vital structures. The proximal end was bluntly dissected to a broad, sessile attachment in the area of the genial tubercles. Vessels leading to the tumor mass were ligated and cut at that point. Considerable effort was required to dissect the tumor from its attachment at

the base. A Penrose type of drain was placed to prevent hematoma formation, and the area was closed with deep gut and superficial silk interrupted sutures. The patient tolerated the procedure well and left the operating room in good condition.

Postoperative course: The patient did well postoperatively. He was seen daily by the oral surgery service. The drain was removed on the second postoperative day, the sutures were removed five days later, and the patient was discharged on June 12, 1959.

Pathologist's Report.—The gross specimen consisted of two portions, both composed of fatty tissues. The larger measured up to 7 by 6 by 3 cm. In this larger portion of tissue there were also muscle, tendon, and a portion of bone. The bony portion measured 2 by 1.2 by 1 cm.

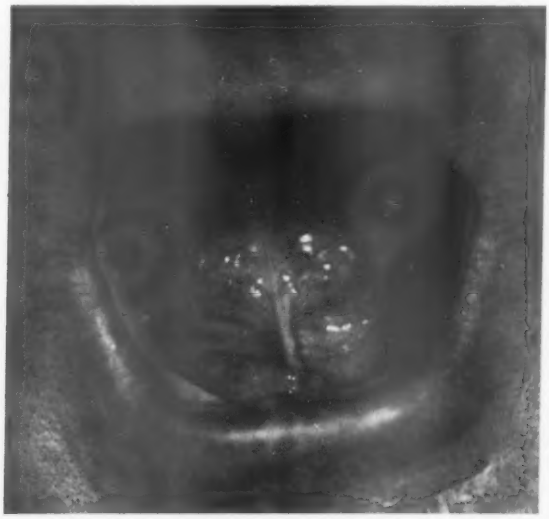

Fig. 4.—Postoperative photograph taken seven months after removal of the tumor. The tongue now rests in its normal position, the submaxillary ducts are patent, and there is no evidence of residual or recurrent tumor.

Microscopic sections showed well-formed cancellous bone surrounding striated muscle, fibrous connective tissue, and fat. One section showed adult adipose tissue.

Diagnosis: Lipoma of the floor of the mouth with ectopic bone formation.

SUMMARY

A case of lipoma originating adjacent to the mandibular periosteum in the area of the genial tubercles is presented. Within the tumor, and attached to a common base, was an osseous projection whose general shape and direction of growth corresponded to the fatty tumor. In reviewing the literature, we found no report of a lesion with the same morphology.

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"TRUE" (ATTACHED) CEMENTOMA WITH ROOT CANAL INVOLVEMENT

Report of a Case

F. W. Wertheimer, D.D.S., M.S.D., M.S., E. J. Driscoll, D.D.S., and H. R. Stanley, B.S., D.D.S., M.S., Bethesda, Md.

ALTHOUGH no attempt will be made to review the literature on cementomas, it may be beneficial to discuss briefly the terminology that has been applied to this class of lesions.

Shafer and his colleagues[1] characterized a "true" cementoma as a neoplasm of cementum attached to the tooth root and distinguished from hypercementosis by its great bulk of calcified tissue, its tendency to expand the bony plates, and its active appearance histologically.

Thoma and Goldman[2] believe that cementomas can be attached or unattached and that they arise from odontogenic fibromas derived from the dental follicle or its end product, the periodontal membrane. They use the terms "cementoblastoma" when only a small amount of calcified tissue is present and "cementoma" when the lesion is principally cementum.

Bernier[3] preferred to reserve the term "cementoma" for accumulations which are attached to the teeth, calling the unattached lesions "cementifying fibromas."

Unless the calcified material is attached or continuous with the tooth cementum, it is sometimes difficult to determine whether it is, in fact, cementum or bone. Although Bernier recognized certain histologic criteria as diagnostic for cementum, Weinmann and Sicher[4] claimed that cementum could not be recognized as a specific tissue per se, and stated ". . . only a mass of hard tissue continuous with the normal cementum should be called cementoma."

Although numerous reports of so-called cementomas have appeared in the literature, few could satisfy Shafer's criteria for a "true" cementoma. Probably many of the unattached types could be classified as cementifying or ossifying fibromas (odontogenic fibromas or cementoblastomas of Thoma), fibro-osteomas, fibro-osteoid osteomas, or osteomas. To be less specific, the term "periapical fibrous dysplasia" also has been suggested for the unattached

From the Clinical Investigations Branch, National Institute of Dental Research, United States Public Health Service, Department of Health, Education, and Welfare.

lesions.[5] Examples of these unattached lesions were usually static and symptomless, being discovered by means of routine dental radiographs, and required no treatment unless symptoms developed.

The "true" cementoma, by contrast, is apparently a comparatively rare lesion and usually should be removed because of its active growth and because of the symptoms produced. No statistics on the incidence of "true" attached cementomas have been found, since all of these lesions, whether attached or unattached, have been grouped together in past surveys.

The only reference that we found to root canal involvement in a lesion of this type was in an article by Blum,[6] who discussed a case of hypercementosis or cementoma that exhibited slight invasion of the apical root canal.

The case that follows satisfies all criteria for a "true" attached cementoma and, in addition, exhibits marked involvement of one root canal.

Fig. 1.—Gross specimen showing tumor mass attached to lingual side of maxillary right second and first molars.

CASE REPORT

The patient was a 14-year-old white boy whose immediate complaint was soreness of the gingivae in the region of the maxillary right second molar. The patient's physician had diagnosed the condition as a growing third molar. A subsequent dental examination disclosed a palatal swelling extending to the midline in the region of the maxillary right second molar. Slight buccal swelling was also noted. The patient claimed that the bony swelling was of approximately four months' duration. Radiographically a large, circumscribed, radiopaque area was visualized involving the roots of the maxillary right first and second molars.

An attempt was made to obtain a biopsy specimen of the bony lesion which was judged to be under the second molar. It was impossible to move the second molar under any circumstances, even after a wide buccal flap had been made. Therefore, the first molar was removed without difficulty and the osseous lesion was dissected and removed in toto.

The bonelike mass encased the roots of the second molar, with some fragments adherent to the first molar (Fig. 1). It measured 2.5 cm. in diameter and was brownish gray

in color. Microscopic examination revealed a large, bonelike mass, continuous with the cementum of the tooth, enclosed in a fibrous connective-tissue capsule (Figs. 2, 3, and 4). The bulk of the tissue was composed of nests, islands, and trabeculae of bonelike substance with fibrous connective-tissue stroma. Numerous osteoblasts and osteoclasts were present

Fig. 2.

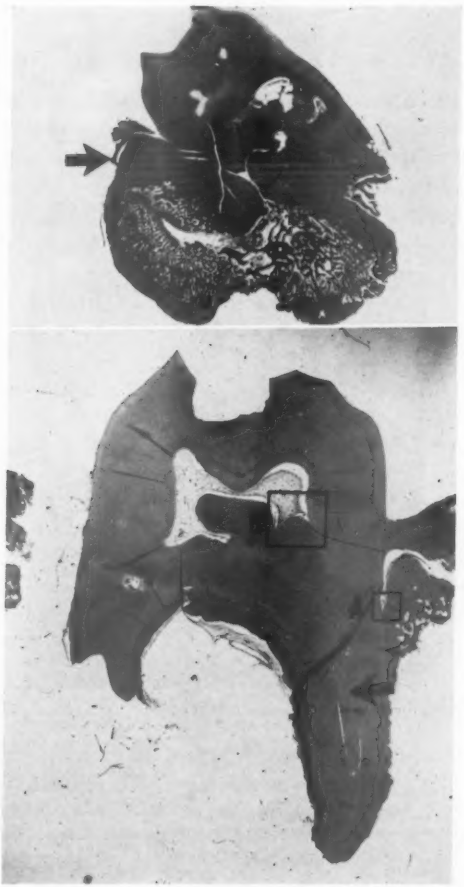

Fig. 3.

Fig. 2.—Photomicrograph of second molar with tumor attached. Buccolingual section. (Magnification, ×2; reduced ⅙.) Box indicates area shown in Fig. 4.

Fig. 3.—Photomicrograph of first molar, buccolingual section. Arrow indicates root canal filled with cementum-like tissue. (Magnification, ×5; reduced ⅙.)

around the periphery of the calcified tissue. In one area of periodontal membrane, at the edge of the lesion near the gingival crest, cementoid formation and thickening of the cementum blending into the main bulk of the tumor could be seen (Fig. 5). An interesting feature was the apparent invasion or formation of bonelike substance within the lingual root canal of the first molar (Fig. 3). Serial sections were made and the presence of the

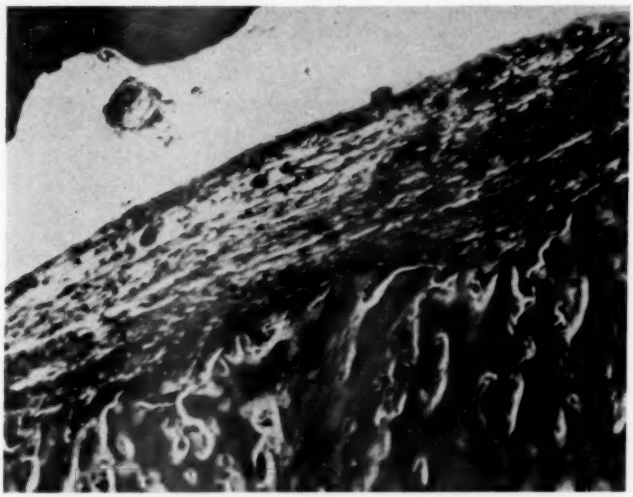

Fig. 4.—High-power view of boxed area in Fig. 2 showing fibrous connective-tissue capsule on periphery of tumor mass. (Magnification, ×130; reduced ⅙.)

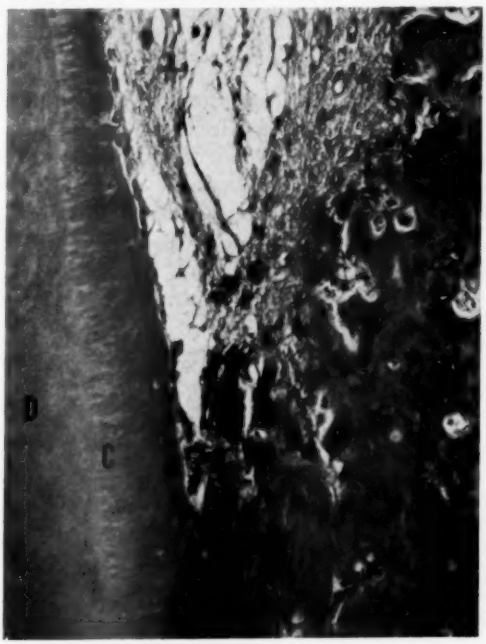

Fig. 5.—High-power view of area in box *A*, Fig. 3, showing blending of tumor mass with cementum of tooth and apparent formation in periodontal membrane. *C*, Cementum; *D*, dentine. (Magnification, ×210; reduced ⅙.)

calcified substance throughout the root canal, extending to the level of the base of the pulp chamber, was confirmed. It was separated from normal pulp in this area by a layer of cementoblasts (Fig. 6).

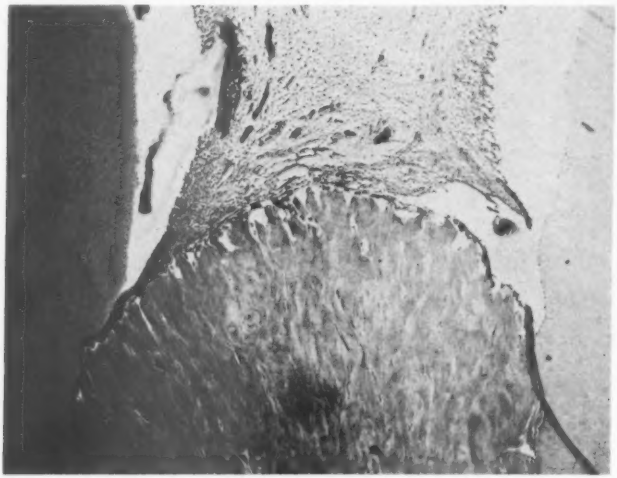

Fig. 6.—High-power view of area in box *B*, Fig. 3, showing junction of tumor mass and pulp tissue in pulp chamber. (Magnification, ×150; reduced ⅙.)

DISCUSSION

Endless arguments of an academic nature have taken place over the proper terminology for this group of periapical lesions. The cell of origin is the undifferentiated mesenchymal cell which can become a fibroblast, an osteoblast, a cementoblast, or an odontoblast. In the case of cementum and bone, which cannot always be differentiated on a morphologic basis, the environment must be considered in the making of a diagnosis. Because of the clinical history of rather rapid growth, the encapsulation of the lesion, and the blending of the tumor with the cementum of the tooth, the lesion in the case just presented was considered an example of a "true" (attached) cementoma, denoting neoplasia. This case was considered especially interesting since "true" cementomas with root canal involvement are apparently rare.

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ANNOUNCEMENTS

American Academy of Dental Medicine

The Academy will hold a cruise convention aboard the luxury liner *S. S. Ocean Monarch* to Bermuda and Nassau over the Memorial Day week end, May 26 to June 3, 1961, combining a comprehensive scientific session with a program of recreation.

The theme of the scientific program is "Recent Trends in Dental Medicine." Lectures will be given by Hermann Becks, Professor of Oral Biology, University of California School of Dentistry; J. Lewis Blass, Professor of Periodontia and Oral Medicine, New York University College of Dentistry; and Milton Hyman, Professor of Oral Diagnosis and Roentgenology, New York University College of Dentistry. Two symposiums will be conducted by members of the various sections of the Academy.

While in Bermuda members of the Academy will be guests of the Bermuda Dental Society at a buffet dinner–lecture. Dr. Hermann Becks, President of the Academy, will address the joint meeting.

Superb service and cuisine, nightly dancing and entertainment, sight-seeing, and sports activities will be included in the recreational program.

All interested dentists and physicians, as well as Academy members and their families, are welcome. For details and reservations, address Dr. Howard Ward, General Chairman, 15 Bond St., Great Neck, New York.

Twelfth Annual Berkshire Conference in Periodontology and Oral Pathology

Major General Joseph L. Bernier, Assistant Surgeon General and Chief of the Dental Corps, United States Army, will be one of the principal speakers at the Conference, which will be conducted at Eastover in Lenox, Massachusetts, June 18 to 22, 1961. Other lecturers on the Conference faculty will be Finn Brudevold, Irving Glickman, Reidar F. Sognnaes, John B. MacDonald, Sigurd Ramfjord, Everett Shapiro, David Weisberger, and Frank M. Wentz.

The Conference is open to general practitioners and dentists in specialized practice. Information may be obtained from Tufts Berkshire Conference, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

Dental Internship Available at Hospital for Joint Diseases

The hospital announces that a position is available for a rotating dental intern in its approved Department of Dentistry for the year beginning July 1, 1961. The department is headed by Dr. Stanley J. Behrman, Attending in Dentistry, and the training includes oral surgery, orthodontics, oral pathology, radiology, and periodontics.

The hospital provides its interns with room and uniforms, plus a cash stipend of $110.00 a month and insurance coverage.

Applications should be addressed to Executive Director, Hospital for Joint Diseases, 1919 Madison Ave., New York 35, New York.

New York Institute of Clinical Oral Pathology, Inc.

A course in clinical oral pathology is offered on two Wednesday evenings a month, October through May (sixteen sessions). The course includes lectures on embryology, hematology, histology, and pathology (illustrated by lantern slides, blackboard demonstration, microscopic slides, etc.) and their clinical interpretation. In addition, case reports stressing history, clinical findings, x-ray studies, diagnosis, treatment (including diagnostic and surgical procedures), histopathologic evidence, and prognosis will be given by the staff of the Institute and by guest speakers. The course is approved by the Board of Education of the University of the State of New York.

Inquiries should be addressed to the executive secretary, 101 East 79th St., New York 21, New York.

American Society of Dentistry for Children

The Society will hold its annual meeting Oct. 13 to 15, 1961, at the Bellevue-Stratford Hotel in Philadelphia, Pennsylvania. The scientific sessions will include a closed-circuit television program on cleft palate treatment, a panel discussion on growth patterns in children, and table clinics. In addition, there will be a full social program.

For further information, write A. S. D. C. Convention Director, Medical Arts Building, York Road and Hillside Avenue, Jenkintown, Pennsylvania.

The Academy of General Dentistry

On Wednesday, May 10, 1961, the Dental Department of Michael Reese Hospital and Medical Center will present an all-day course on "The Hospital—An Adjunct to Dental Practice" expressly for members of The Academy of General Dentistry.

For full details and the complete program, write to Dental Department, Michael Reese Hospital and Medical Center, 29th and Ellis Ave., Chicago 16, Illinois.

Albert Einstein College of Medicine, Yeshiva University

The Department of Dentistry will sponsor a five-day participation course in clinical periodontics, Monday to Friday, Oct. 30 to Nov. 3, 1961. Each participant will have an opportunity to perform curettage, temporary splinting, and a simplified surgical procedure. This course is basically designed for out-of-town practitioners. Tuition is $175.00. The course is under the direction of Drs. Irving Yudkoff and Marvin N. Okun, and enrollment is limited.

For further information, please write Director, Postgraduate Dental Program, Albert Einstein College of Medicine, Eastchester Rd. and Morris Park Ave., Bronx 61, New York.

Oral Surgery Residency at Mount Sinai Hospital

A two-year approved residency in oral surgery has been introduced at the Mount Sinai Hospital in New York City. Both didactic and clinical training of the required types are included.

Information and application forms may be obtained from Dr. Alvin Nathan, Department of Dental and Oral Surgery, the Mount Sinai Hospital, Fifth Ave. and 100th St., New York 29, New York.

Postgraduate Courses at Mount Sinai Hospital

The following postgraduate courses for the general practitioner will be offered in the autumn of 1961:

PG-61/1. Analgesia and Pain Control.

PG-61/21. Minor Oral Surgery for the General Practitioner.

For further information, catalogue, and application forms, write to Dr. A. Norman Cranin, Chairman, Education Committee, Department of Dental and Oral Surgery, The Mount Sinai Hospital, Fifth Ave. and 100th St., New York 29, New York.

University of Pennsylvania

The School of Dentistry offers the following postgraduate courses:

Principles of Occlusion. May 3 to 5, 1961. Lewis Fox, Jack Alloy, Morton Amsterdam, Walter Cohen, Leonard Abramson, and Morris Feder.

The Use of Ultrasonics in Periodontia. May 6, 1961. Henry M. Goldman and D. Walter Cohen.

Biologic Aspects of Endodontics. May 22 to 26, 1961. Samuel Seltzer and I. B. Bender.

Endodontics—Immediate Root Resection. June 5 to 10, 1961. Louis I. Grossman and associates.

The Twin-Wire Mechanism. June 5 to 9, 1961. Joseph E. Johnson.

Orthodontics for the General Practitioner. July 10 to 14, 1961. Robert E. Moyers and associates.

On Saturday, May 13, 1961, Drs. Henry M. Goldman, D. Walter Cohen, Morton Amsterdam, and Lewis Fox will present a one-day seminar for those who have attended a postgraduate course in periodontal therapy at the School of Dentistry, University of Pennsylvania, or at Beth Israel Hospital, Boston, Massachusetts.

On Thursday, May 18, 1961, Dr. M. M. DeVan will present a one-day seminar for dentists who have taken his postgraduate course in complete denture prosthesis.

For further information, write Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

Boston University School of Medicine

The Oral Surgery Section offers a one-year oral surgery course planned to meet the requirements of the American Board of Oral Surgery. It provides a foundation for clinical training, such as clinical internship and residency, leading to qualification for the practice of oral surgery as a specialty. This course, given annually, is designed to give, by lecture, clinical demonstration, operative clinic, and laboratory exercise, a systematic review of the clinical phases of oral surgery. This is coordinated and correlated with the pertinent preclinical sciences. The fundamental principles of diagnosis and treatment of the various manifestations in the field of oral surgery are covered, with specific emphasis on etiology and pathology. Instruction is given in oral surgery, principles of general surgery, animal surgery, radiology, general and oral pathology, oral medicine, physical diagnosis, medicine, dermatology, clinical laboratory procedures, anesthesia, physiology, biochemistry, pharmacology, anatomy (head and neck), bacteriology, surgical prosthesis, and oncology. The course is under the direction of Dr. Kurt H. Thoma, Professor of Oral Surgery.

The Orthodontics Section offers a course of two academic years intended for those who wish to prepare themselves for the practice of orthodontics as a specialty. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of the specialty. Courses are given in the preclinical sciences pertaining to this field. Applications are being accepted for the program which begins in the fall of 1962. The course is under the direction of Dr. Herbert I. Margolis, Professor of Orthodontics.

The Prosthetics Section offers a one- or two-year course in general prosthetic dentistry. It is intended for those who wish to prepare themselves for the practice of prosthodontics as a specialty and/or who may wish to prepare for the specialty board examinations. This course consists of lectures, clinical demonstrations, operative clinics, and laboratory exercises

in all clinical phases of the field. These are coordinated and correlated with pertinent preclinical sciences. The course is under the direction of Dr. Leo Talkov, Assistant Professor of Prosthetic Dentistry.

The Endodontics Section offers a one-year course to dentists wishing to specialize in the field of endodontics. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of this specialty. Instruction is given in the preclinical sciences pertaining to this field. The course is under the direction of Dr. Herbert Schilder, Assistant Professor of Endodontics.

The Oral Pathology Section offers a two-year course intended to prepare dentists for teaching or research in this field. The program includes the pertinent basic sciences and clinical oral medicine. The related curriculum consists of anatomy, biochemistry, physiology, microbiology, oncology, dermatology, general pathology, and oral pathology. The general pathology is given in connection with the Pathology Department of the Massachusetts Memorial Hospitals.

For application or information, please apply to Department of Stomatology, Boston University School of Medicine, 80 East Concord St., Boston 18, Massachusetts.

Balint Orban Memorial Seminar

The Seminar (formerly known as the Rocky Mountain Dental Seminar) will be held in Boulder, Colorado, June 25 to 28, 1961. The theme for the Seminar is "Clinical and Microscopic Pathology of the Periodontium and Other Oral Structures." The outstanding group of essayists includes Drs. S. N. Bhaskar, Henry Goldman, Maynard K. Hine, and Harry Sicher.

Further details and application may be obtained by writing to Mrs. James Manella, Secretary, Balint Orban Memorial Seminar, 1700 Mesa Rd., Colorado Springs, Colorado.

Michael Reese Hospital and Medical Center

July 1, 1961, will mark the close of the first year of successful operation of the Mandel Clinic interceptive orthodontic service. Dr. Max Gratzinger, who for many years was associated with Dr. A. Martin Schwarz in Vienna, supervised the clinic and directed the teaching.

Because of the rewarding results and the satisfaction expressed by participating dentists, the administration has decided to continue the clinic and to make the course again available to dentists engaged in general practice.

Dentists interested in participating in this exceptional learning experience should write to the Dental Department, Michael Reese Hospital and Medical Center, 29th and Ellis Ave., Chicago 16, Illinois.

REVIEWS OF THE LITERATURE

BOOK REVIEW

Dentistry for the Pre-School Child. C. N. Davis and R. M. King., University of Otago Dental School, Dunedin, New Zealand. Baltimore, 1961, Williams & Wilkins Company (exclusive U. S. agents). Price, $7.50.

The authors place emphasis upon principles rather than techniques. "It is relatively easy to determine the nature of a child's dental problem; it is both more difficult and more important to determine why the child has that problem."

There are fifteen chapters, including some on the management of children and on childhood diseases which have oral manifestations or which influence dental treatment. Each chapter has a complete bibliography.

E. Parnes.

A Handbook of Differential Oral Diagnosis. Major M. Ash, Jr., Associate Professor of Oral Pathology and Periodontia, The University of Michigan School of Dentistry. St. Louis, 1961, The C. V. Mosby Company. Price, $6.50.

This short monograph is not intended to be all-inclusive, according to the author; rather, it is "a brief and practical index to the oral manifestations of disease."

The topics discussed are arranged alphabetically (A, abrasion; B, bleeding; C, cysts; L, leukemia; V, vitamins, etc.). It is nicely arranged for quick reference.

I know of no other pocket-size book of this general nature. This one will be useful, but it is regrettable that the author chose to exclude illustrations and references.

When a summary of the salient facts is required immediately prior to examinations, the student can find them in these pages.

Thomas J. Cook.

ABSTRACTS OF CURRENT LITERATURE

The Histopathology of Masseteric Hypertrophy. Paul Guggenheim and Leon B. Cohen. A. M. A. Arch. Otolaryng. 71: 906 June, 1960.

The case of a patient with long-standing chronic unilateral hypertrophy of the masseter muscle is reported. Biopsy disclosed small isolated islands of cartilage in the masseter specimens. This finding is believed to represent an associated myositis ossificans caused by the trauma of recurring painful attacks of masseter spasm and swelling.

Hereditary Benign Intraepithelial Dyskeratosis. C. J. Witkop, C. H. Shankle, J. B. Graham, M. R. Murray, D. L. Rucknagel, and B. H. Byerly. A. M. A. Arch. Path. 70: 696, December, 1960.

This report describes the oral lesions of a hereditary congenital syndrome characterized by benign intraepithelial dyskeratosis affecting the oral mucosa and bulbar conjunctiva. The oral lesion affects the buccal and labial mucosa, the floor of the mouth, and the sides of the tongue.

Oral examinations were made of 540 persons, including 345 of the 582 living descendants of a woman born in 1834. They were members of a triracial isolate residing in Halifax County, North Carolina. The oral condition was entirely asymptomatic. Children were lightly involved, and the condition increased in severity with age up to 15 years. The earliest case reported was that of a 6-week-old boy whose symptoms were first diagnosed as thrush.

The lesions varied in clinical appearance with the severity of involvement. In slightly affected persons, the buccal mucosa was white, spongy, and macerated. When the mucosa was scraped to remove the superficial cells and then stretched, the surface appeared to be covered with pin-point projections that looked like white opaque plastic points.

Biopsy specimens of the mucosa and scrapings stained by the Papanicolaou technique were studied to confirm the diagnosis of intraepithelial dyskeratosis.

The condition is benign, and it is interesting that no one showed involvement of the tongue, pharynx, or uvula.

The article is well written; it has colored illustrations and excellent references. It is recommended to the dentist, the specialist, and the general practitioner.

Cartilage Pharyngoplasty in Cleft Palate Patients. R. F. Hagerty and M. J. Hill. Surg. Gynec. & Obst. 112: 350, March, 1961.

In the cleft palate patient who has a speech difficulty, a rather short palate with a limited range of motion, coupled with a rather large nasopharynx, is frequently found. Even after surgical repair of the cleft palate, these same characteristics often combine to prevent satisfactory speech. The surgical approach to improve or correct these anatomic abnormalities can be considered under three general headings: (1) displacing the soft palate posteriorly, (2) attaching the soft palate to the posterior pharyngeal wall, and (3) displacing the posterior pharyngeal wall anteriorly. This study is concerned primarily with procedures designed to move the posterior pharyngeal wall forward and thus improve velopharyngeal closure.

Viable human cartilage grafts—two autogenous and the remainder homogenous—were placed in the posterior pharyngeal walls of twenty postoperative cleft palate patients. A transoral approach was used in order to reduce the velopharyngeal insufficiency. These patients were studied preoperatively and one month and one year postoperatively by means of upright cephalometric laminagraphic roentgenograms and speech recordings. There was almost complete loss of four such grafts, one of which was autogenous. In general, there was satisfactory maintenance of the pharyngeal wall projection by three-fourths of the grafts after one year of transplantation. The transoral approach was more satisfactory than the transcervical approach in permitting a sufficiently high placement of the grafts.